

PULMONARY TUBERCULOSIS IN PAUPER PATIENTS, WITH.

SUGGESTIONS FOR THEIR CURE AND TREATMENT.

Being a Thesis presented

by

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The following Thesis is based on eighty consecutive cases of Pulmonary Tuberculosis admitted to the Bradford Workhouse Hospital. It deals more particularly with the relation of other diseases to Pulmonary Tuberculosis and with the treatment adopted; with added notes on Phthisical patients under the Poor Law.

#### ETIOLOGY:

Considering the wide distribution of the Tubercle bacillus and the many chances for infection, it seems wonderful that in spite of it so many persons escape the diseases produced by it.

One factor as pointed out by Koch must be borne in mind, however, and that is the extremely slow growth of the organism, hence the bacilli may be eliminated before they have gained a definite foothold.

Individual predisposition is another factor which is still more important - a factor not readily explainable - but one which we cannot very well get on without. Only a part fall ill, of all who are exposed to the organism.

There are persons in whom it is particularly easy for the organism to establish and propagate itself. This inborn predisposition of the individual/

idual is no doubt the strongest factor in the etiology of the disease.

In these eighty cases the difficulty in obtaining any evidence of a hereditary tendency was very great. Only in a minority of the cases was it possible to obtain a history of tuberculous disease, such as Scrofulous glands, disease of the Bones or Joints, or Pleurisy, - in the family.

The cases under consideration are drawn from the poorest class; the majority are drunkards, living in overcrowded and ill-ventilated common lodging houses and frequently having insufficient food; hence it is that the alcoholic - once perhaps of strong constitution - offers a feeble resistance to Tuberculosis, so that the rapidly spreading forms of Pulmonary Tuberculosis appear to be particularly common in them.

#### PREVIOUS HEALTH:

Very little reliance can be placed upon the statements made concerning the previous health. A history of Bronchitis, Influenza, Pleurisy and Pneumonia was frequently obtained.

Bronchitis, owing to frequent exposure, is common amongst patients admitted into Workhouse Hospitals, but I doubt if it in any way predisposes to Pulmonary Tuberculosis.

A history of Influenza was frequently given; it is not uncommonly followed by Phthisis.

Pleurisy, in like manner, is liable to be followed after a longer or shorter interval by it. With Pneumonia, however, it is doubtful. There is no evidence to support the belief that it is followed by Pulmonary Tuberculosis. On the other hand, many of the cases which were supposed to be a Simple Pneumonia at the beginning, were probably tubercular from the outset.

OCCUPATION:

The eighty cases were distributed as follows:-

Labourers .....	24
Hawkers .....	23
Millworkers .....	15
<i>Local</i> <del>Hay</del> sorters .....	8
Tailors .....	5
Cabdrivers .....	4
Waiters .....	3
Bakers .....	2
Quarrymen .....	2
Enginefitters .....	1
Domestic Servants .....	<u>3</u>
	80

Thus the majority were engaged at work involving much exposure to all kinds of weather or to the inhalation of dust.

SEX AND AGE:

Dr Ogle calculating from the death rates, finds/

finds the mortality over the whole of life in the two sexes is the same, but that at the different age periods, the rates for the two sexes differ considerably. Before 35, it is higher for females, after 35 the rate is higher for males and remains so. Eight females were over, and 12 males under 35 years of age.

yrs.	10-20	- 30	- 40	- 50	- 60	- 70	- 80
F.	4	10	10	3	0	0	1
M.	3	6	11	19	7	4	2

The frequency in males and females at different age periods is said to be due to the strain of life falling heavily on woman during the child-bearing period; it falls upon men at a later period.

Not only those with a predisposition to the disease fall ill, but also healthy individuals who, by vitiating influences such as bad hygiene, insufficient food and alcoholism, lower their natural powers of resistance. Probably the presence or absence of this inherent tendency decides the acuteness of the lesion. Of predisposing factors, alcohol, I believe, is the most powerful.

MODE OF ONSET:

1. Bronchitic. Fifty per cent. of the cases began with symptoms of bronchitis. The history given, was that for many winters the patient had had a cough, but the present attack had persisted and was getting worse.
2. Pleuritic type in which the illness began with stitchlike pains accompanied by cough and fever; 22.5% had such an onset.
3. Insidious, in which there was loss of flesh and strength, or complaint of digestive disturbance; 20% of the cases had such a beginning.
4. *Hæmoptic* in which the first symptom taken notice of by the patient is spitting of blood. This form occurred in 2.5% of the cases.
5. Laryngeal form: The patient complains of hoarseness and loss of voice; 5% of the cases had such an onset.

CLINICAL TYPES:

I have followed the classification adopted by Kinghorn ~~Kinghorn~~ Fowler, viz.,

1. Acute Miliary Tuberculosis of the lung.
2. Caseous.
3. Fibro-Caseous.

## 4. Fibroid or Chronic.

With the exception of Acute Miliary Tuberculosis, I do not propose to consider clinically the other forms.

Of the total number of cases (80), to of them were of the first type.

CASE 1:

T.V., 42, male, by occupation a ~~coal~~comber admitted under a lunacy order on the 28th of August, 1902, and said to be suffering from Mania and Influenza.

His friends stated that he had been ill for three weeks previous to admission. His illness began gradually with a feeling of out of sorts, headache and short irritable cough. He steadily got worse and on the day of admission, he became delirious and had to be placed under control. He had been in comfortable circumstances and was well fed. There was no history of intemperance, his family history was good and his previous health satisfactory. On admission, the patient appeared to be very ill. He was extremely emaciated, his face was pinched and deeply cyanosed and his eyes bright/



P.M. The pleurae were healthy. Throughout both lungs were numerous tubercles the size of a hemp seed. Both lungs were oedematous and congested. At the right apex, there was an old tuberculous focus the size of a marble.

The Spleen and Kidneys showed numerous minute tubercles. The other organs appeared to be healthy.

#### CASE II.

E.V., 49, tailoress, admitted on the 30th September 1902. For the previous sixteen weeks she had been troubled with a short cough and dull aching on the left side.

Four months previously, patient was in the Royal Infirmary and whilst there, four pints of fluid had been aspirated from left pleural cavity. Since then she had been in indifferent health and had a sharp pain on left side and a cough. She suffered from attacks of faintness and palpitation from time to time. She steadily got worse and was compelled to give up work. This she did 3 days before admission.

On admission, her temperature was 103.8°F., respirations 32 and pulse 100, regular and of poor tension.

She/

She was emaciated but well developed. Her face was flushed and slightly cyanosed. Venules on cheeks dilated. Her eyes were bright and watery and expression rather anxious. She had a short dry cough but no expectoration.

Examination of chest revealed slight impairment of front of left lung with diminished vocal fremitus and resonance. Breathing of vesicular type rather faint and jerky. There were no accompaniments, posteriorly as in front, but resonance less good. Breath sounds were very faint at the base, with a few sharp crepitations.

The cardiac dulness was increased, the right border of the heart being  $\frac{1}{2}$ " from Right Sternal margin and the left border  $\frac{1}{4}$ " outside the nipple line. Over the proecordium, there was a rough systolic murmur. It replaced the first sound at the apex and was transmitted as far as the left anterior axillary line.

The murmur was loudest and appeared very superficial at the tricuspid area, but did not replace the first sound. It was heard also at the base and was conducted along vessels and down the sternum.

The tongue was slightly furred. The Liver and Spleen were not increased. The urine contained/  
ed/

ed a trace of albumen.

On 4th October (4 days after admission) the following note was made. Patient complains of headache and has vomited twice. There are more crepitations at left base and a few at the right.

7th October: Patient appears to be very ill. She is dull and apathetic and slightly delirious. Somewhat deaf for the past 3 days. Tongue thickly furred, sordes on teeth. Pulse regular but dicrotic. Numerous crepitations over left lung. The systolic murmur appears very superficial. The Spleen is enlarged and its edge readily felt.

11th October: Has vomited twice. Slight diarrhoea; stools have a typical pea soup character. Face flushed, cyanosis very marked, breathing rapid - 32 per minute and somewhat laboured. Perspiring profusely. Systolic murmur much fainter. Crepitations very numerous over left lung and expiration prolonged and louder than inspiration.

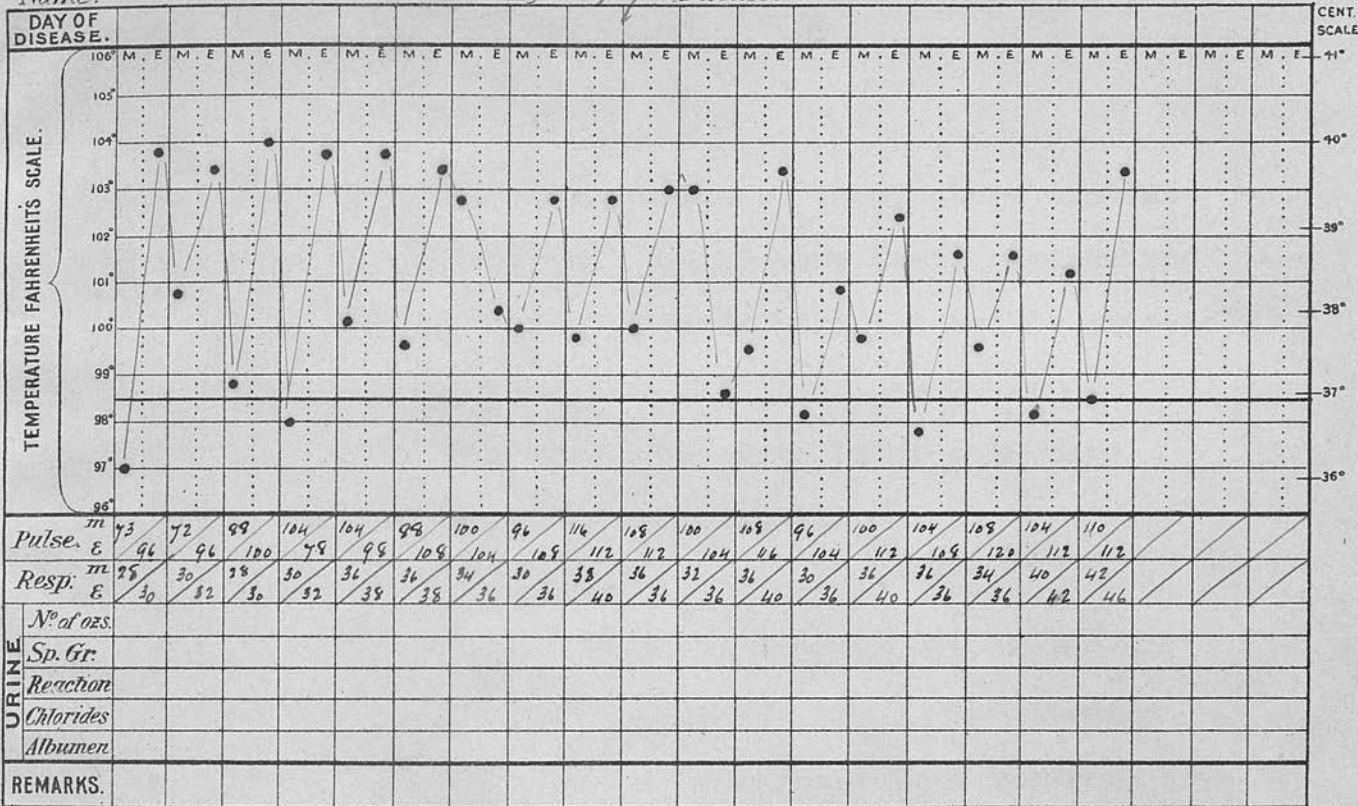
15th October: Sweating profusely, Miliaria Alba especially on the chest. Very cyanosed, breathing hurried - 36 per minute. Crepitations numerous over both lungs, otherwise no change in the lung condition. No murmur heard over heart. Abdomen/

Name.

E. V.

Age. 49. Disease.

Result.



(Observations taken at ..... A.M. and ..... P.M.)  
 For Memoranda of Treatment, see back of Chart.

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Abdomen a little distended. Temperature continuous, 102° - 104° F. Pulse very fast, regular and dicrotic. Patient appears very ill and is extremely weak.

Death occurred three days later - 18 days after admission. (vide Chart).

P.M.

The left pleura was thickened but showed no evidence of Tubercles. Both lungs were studded with Miliary Tubercles. There was no evidence of an old tuberculous lesion anywhere in the lungs. A few milk spots on left ventricle. No percarditis.

Numerous tubercles in both kidneys.

REMARKS:

In the first case, I afterwards learned the onset was sudden - in the other it was more gradual, the patient never being in good health since her illness four months previously.

The striking feature in both was the amount of general illness quite out of proportion to the physical signs. Cyanosis in both was very marked; in one it was present from time of admission; in two it was slight and increased as the lung condition/

dition developed; the cheeks were flushed rather than cyanotic, but towards the end <sup>of the case</sup> it was very marked.

Dyspnoea was a striking symptom of both cases, the respirations varying from 28 to 60 per minute.

#### PYREXIA:

No. 1 was not characteristic.

No. 2 was typical.

(See Charts.)

The pulse was feeble in both, dicrotic in No. 2, suggesting pulse of Enteric Fever. In 1, the frequency of the pulse was greater than in 2, whilst in 2, when the temperature was at its highest the pulse was varying from 80-96 per minute, which again suggested Enteric Fever. Cough was only very slight in both cases and there was no expectoration in either. Delirium was present in both, but in none of them very prominent. It was of a low muttering character. Both had profuse perspiration. The Spleen was enlarged in both cases.

The signs in the lungs in either case were very scanty. Crepitations were numerous in both and expiration was prolonged and louder than inspiration.

spiration.

DIAGNOSIS:

The only condition which it seemed likely to mistake No.1 for was Pneumonia of an alcoholic type. Such cases are frequent in Workhouse Hospitals. The patients are often admitted in extremis. The breathing is rapid, cyanosis is marked, and delirium is frequently present. The temperature is quite unlike that of an ordinary Pneumonia in a healthy adult. In Alcoholic Pneumonia, owing to a poor reaction, the temperature remains low, never above 101° - 102°F.

The signs in the lungs are misleading. Consolidation develops only very slowly. What one generally finds is a diffuse bronchitis over both lungs. Expectoration is frequently scanty and not typical.

Leucocytosis is usually absent in these Alcoholic Pneumonias so that examination of the blood is no aid to diagnosis.

No. 1 was of a pneumonic type. The course run by these pneumonic forms of Acute Miliary Tuberculosis shows that the pneumonia is out of the ordinary run. The sweating and dyspnoea and cyanosis/

cyanosis are more prominent. There is no termination by crisis, but the same is seen in Alcoholic pneumonia. The delirium in alcoholic cases is more noisy and violent, and in my experience is oftenest associated with signs at one or other apex. The diminution of Chlorides in pneumonia may be helpful, and of course the discovery of Tubercule Bacilli in the sputum would be decisive, but generally the expectoration is scanty in Acute Miliary Tuberculosis of lungs. The diagnosis in No. 1 was arrived at on account of the marked perspiration, extreme cyanosis and the great disproportion between the physical signs and the general condition of the patient.

The 2nd case presented more difficulties. The history was of some value in directing one's attention to the lungs. The systolic murmur suggested Malignant Endocarditis. In fact, whilst under treatment in the Bradford Royal Infirmary in May, a diagnosis of Malignant Endocarditis was made and Anti-Streptococcal serum given. The murmur, I afterwards learned, was limited to the base and was systolic in time.

At one time, owing to the presence of typical pea soup stools, enlarged spleen, slow pulse, slight/

slight deafness and bronchitis, Enteric Fever was suspected. The absence of typical spots and a negative result on three occasions when the Widal reaction was tried for, were against Typhoid Fever.

After several days the diagnosis became apparent, owing the marked cyanosis, dyspnoea and sweating, and the temperature and the physical signs in the chest.

The case shows the necessity of demonstrating cocci in the blood before administering Anti-Streptococcal Serum. Such a case might readily have been recorded as a case of Malignant Endocarditis successfully treated with serum.

The other cases were distributed as follows:-

Caseous .....	10
Fibro-caseous .....	31
Fibroid or Chronic ....	37.

The lesion in these was distributed in the lungs as follows:-

Right Upper Lobe .....	14
Left " " .....	9
Right Lung (Whole) ....	7
Left " " .....	9
Both lungs, all the lobes, .....	8
Whole of Right Lung and left upper lobe, ....	11
Whole of left lung and right upper lobe, ...	10
Both upper lobes, .....	6
Right Upper and Middle lobes, .....	3
Left base, .....	<u>1</u>
Total	78

GENERAL AND LOCAL SIGNS:

Whilst the lesion manifests itself in the lungs, yet the constitutional disturbance is so great that I think it is well to look on Pulmonary Tuberculosis as a general infection resulting from the production of a toxin or toxins in the lung. We are then in a better position to explain many of the symptoms.

GENERAL SIGNS:

Constitutional disturbance is rarely absent. It may be more marked than the localising signs, consequently it does not stand in any constant relation to the amount of lung mischief. Generally, the constitutional signs are well marked when the disease is active; they are therefore of some value in prognosis.

The constitutional signs are rise of temperature, increased rapidity of the pulse, sweating, loss of flesh and strength and impairment of appetite.

NIGHT SWEATS:

Whilst not peculiar to Pulmonary Tuberculosis, yet no disease shows their occurrence so frequently as it does; 65.5% of my cases gave a history of sweating. It occurred about twice as frequently in the male as in the female sex.

Sweating/

Sweating appears to bear no constant relation to the lung condition. It is certainly more frequent and more profuse in acute and advanced cases, but it is often a very early and distressing symptom in cases in which the lesion is small and not progressing. It seems to have no relation to the fever, for it occurs in chronic apyrexial as well as in pyrexial cases, but it is usually more marked when there is fever. The sweating occurs during sleep, both day and night, so that it might be called more appropriately sleep sweating, rather than night sweating.

It is said to alternate with profuse discharges from other parts and thus cease for the time during profuse diarrhoea (West). In a case of Diabetes Mellitus, sweating was a distressing symptom after the development of Pulmonary Tuberculosis. Yet a large quantity of urine was excreted in this case.

The cause of the sweating has generally been referred to fever and debility, but its relation to fever is inconstant, and occurs in early cases in which there is no exhaustion. More probably it is a toxic symptom and due to stimulation of the secretory nerves or sweat glands by some poison circulating in/  
in/

in the blood. Sudamina occur in Acute Military Tuberculosis, but they appear to be rare in the other forms. The disappearance of sweating is one of the most striking features of the Open Air method of treatment.

#### LOSS OF FLESH:

This may be the first indication of mischief. It is a good measure of the activity of the disease; it is greater or less according to the acuteness of the disease. When the weight becomes stationary, or increases after a period during which there has been a steady loss, a more favourable view of the case may be taken. It generally indicates that the lung condition is quietening down. The loss of flesh may be very rapid and partly may be ascribed to Fever, loss of appetite, profuse sweating and diarrhoea. All of these may be absent and yet loss of flesh is rapid, so we are forced to assume some toxic product as the cause.

A similar rapid loss of flesh occurs in Malignant Disease and that without any involvement of the Alimentary Canal. In this wasting associated with Malignant Disease, some poison must be assumed as the causal agent.

All the spare fat of the body is used up, but curiously enough, the liver is frequently enlarged and fatty. Under favourable conditions, even when the disease is active, weight may be gained. I have at present under observation, a patient who has gained 16 lbs. in 8 weeks. In this case, the temperature ran up to 103° F., and sweating was very profuse and frequent. This rapid increase of weight is more likely to be met with amongst workhouse patients than amongst the well to do.

The pulse and appetite are dealt with under the heading of Circulatory and Alimentary Systems, respectively.

#### LOCALISING SIGNS:

These are the signs which show that the Respiratory Organs are the seat of the disease. They may be divided into:-

1. The Symptoms - cough, expectoration, pain and haemoptysis.
2. Physical Signs.

With the exception of Haemoptysis, I do not propose to discuss the other symptoms nor the physical signs.

HAEMOPTYSIS is a frequent and interesting symptom of Pulmonary Tuberculosis. It may be absent throughout and if it has once occurred, it is likely to be/

to be repeated. Its frequency varies - Wilson Fox's statistics yield a percentage of 72. In my 80 cases, I obtained a history of its occurrence in 56, i.e., 45%. This difference may depend on the type of the lesion. In active forms (caseous) with little or no cavity formation, haemorrhage will occur less frequently. A good proportion of my cases ran a very acute course.

In a small number of cases, haemoph<sup>t</sup>ysis is the first symptom complained of. This mode of onset was seen in 2.5% of my cases. The amount of blood expectorated varies. I have classified my cases as follows:-

Scanty less than 1 oz., .....	21
Moderate, 1-4 oz., .....	12
Profuse above 10 oz., .....	3
	1 fatal.

Only one of the cases ended fatally, so that haemoph<sup>t</sup>ysis cannot be looked on as an urgent symptom; but cases are seen in which the bleeding is small in amount, yet continuous and so after a time may prove fatal from exhaustion.

The haemorrhage is generally small in amount in the earlier cases. Profuse haemoph<sup>t</sup>ysis is associated/  
ted/

ted with chronic cavities and occurs therefore in the older cases.

SEX:

Males ..... 22

Females .....  $\frac{14}{36}$

The greater frequency in the male sex may be explained by the males being exposed oftener to exciting causes and by chronic lesions being commoner in them.

AGE:

Years	10-20	30	40	50	60	70	80
	F. M.	F. M.	F. M.	F. M.	F. M.	F. M.	F. M.
	1 1	4 4	5 7	- 2	- -	- -	1 -

The table agrees with fuller statistics, viz., that haemophysis is most prevalent during early and adult life. In 16 of the 36 cases, it occurred between 35 and 40 years of age. No age is said to be exempt - one of the patients, 71 years of age, had slight haemophysis from time to time.

*hu* The females the average age at which it occurred was 36.7 years; for males, it was 39.3 years.

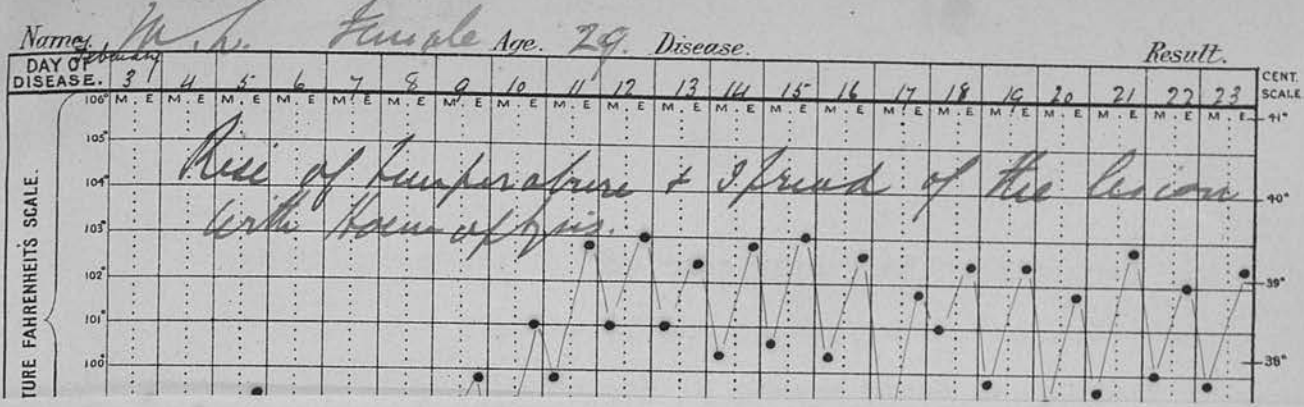
Sometimes/

Sometimes the haemophysis begins abruptly, not infrequently at night without any apparent cause. In others, it is referable to some such cause as bodily exertion, violent paroxysms of coughing, or constipation. A sense of fullness or pain, as West has pointed out, sometimes precedes the haemorrhage. These may be substernal or over one or other lung. This occurrence of pain has a close analogy to the pain preceding a haemorrhage connected with a gastric ulcer or to the pain in the head sometimes experienced before a cerebral haemorrhage.

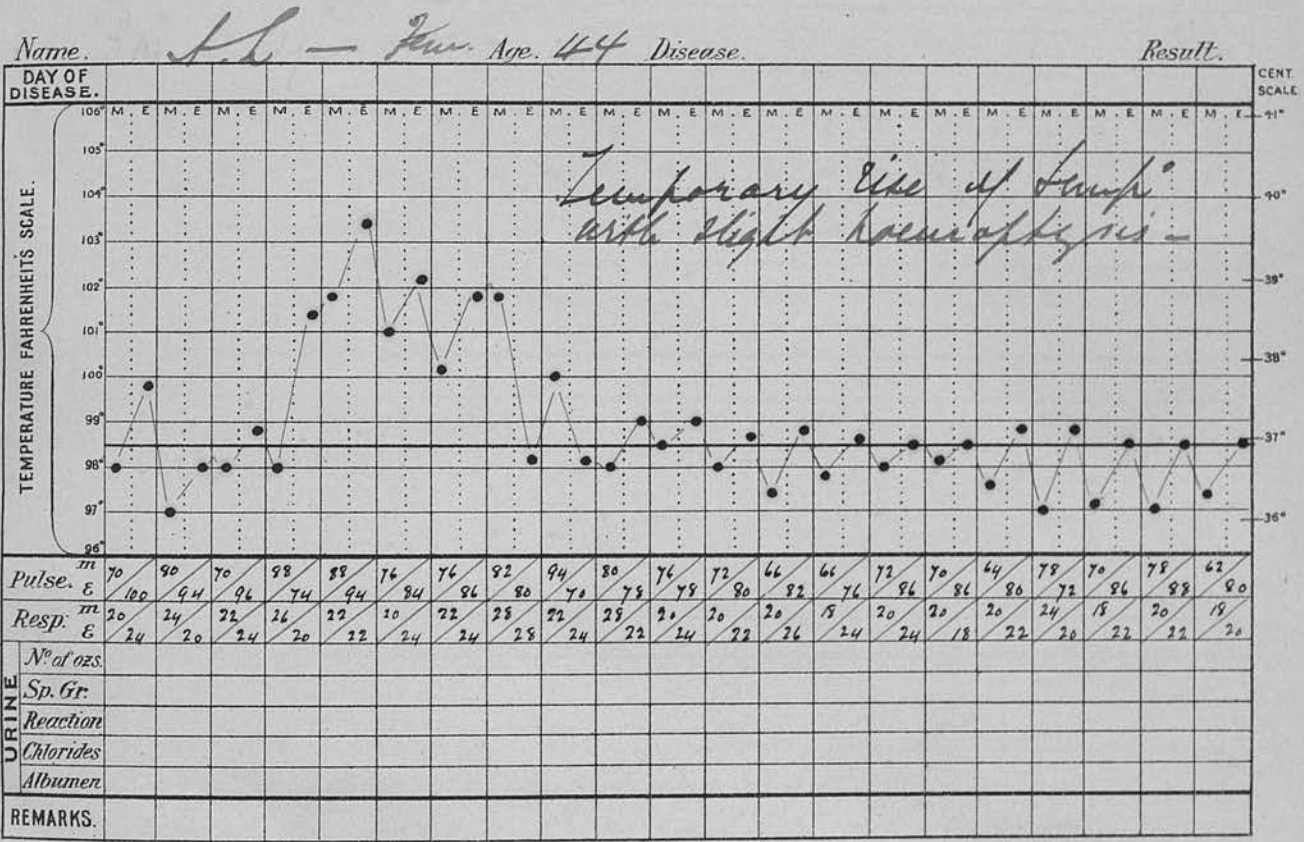
The effect on the patient is variable. He sometimes becomes pale out of proportion to the amount of blood lost; the pulse is full and bounding. This is sometimes due to mental shock, but again it is independent of any excitement on the part of the patient, or even any sort of constitutional disturbance such as fever. Feebleness is an accompaniment sometimes to a degree disproportionate to the amount of blood lost and is an element in the shock which the patient feels at so unexpected an event.

The temperature varies. In some cases it shows no change, in others it may be elevated or become/

TEMPERATURE & C.



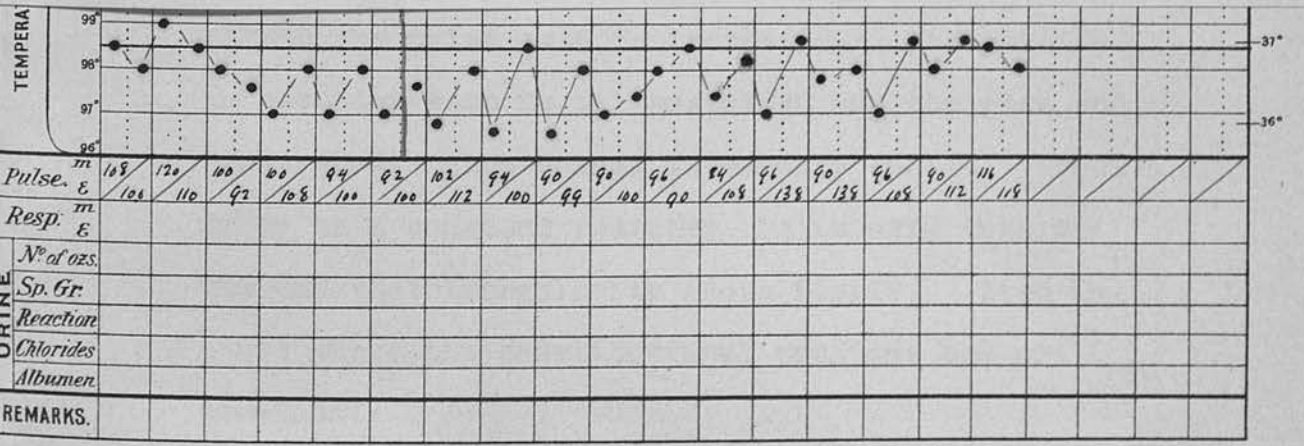
CLINICAL CHART OF TEMPERATURE & C.



(Observations taken at ..... AM and ..... PM.  
For Memoranda of Treatment, see back of Chart.)

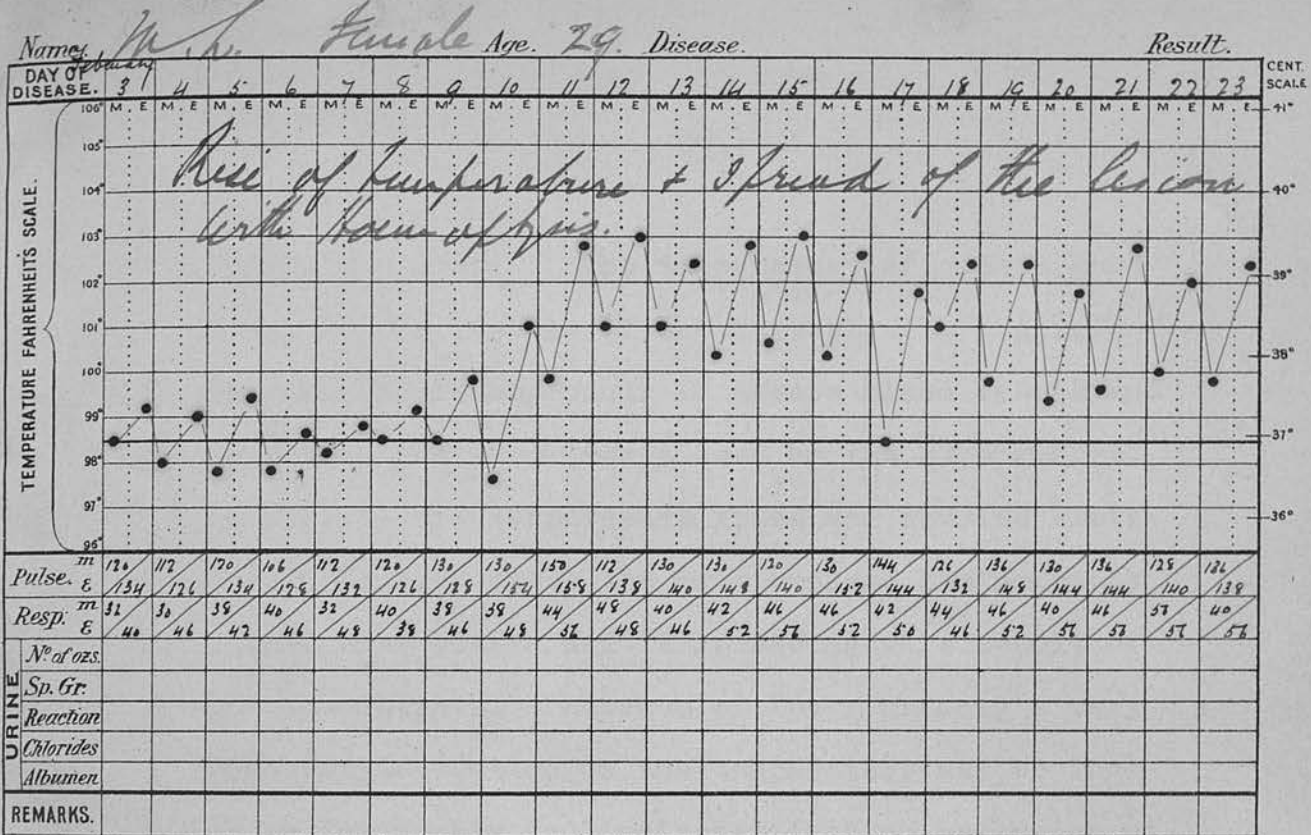
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CLINICAL CHART OF TEMPERATURE & C.



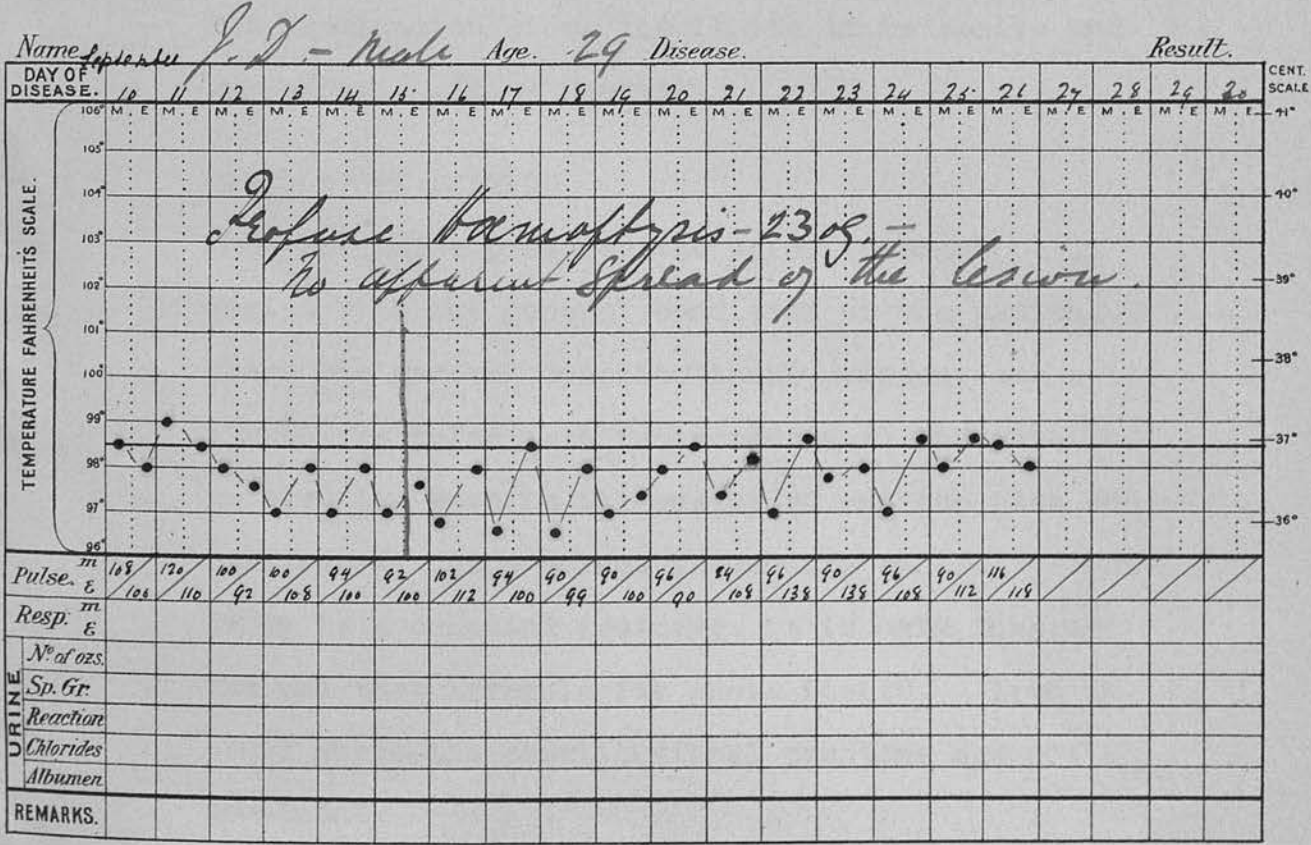
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(Observations taken at AM and PM. For Memoranda of Treatment, see back of Chart.)

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become subnormal. The temperature affords a good guide to the results of the haemoptysis. A fall of temperature may occur in severe blood losses and is due to the shock occasioned by the haemorrhage. In others, the temperature rises and remains elevated. In such cases the lung process has probably become more active and is spreading as a result of the haemoptysis. Absence of fever or only a very temporary elevation of the temperature may be looked on favourably and indicates that no extension of the lung condition has taken place. There are, however, exceptions. There may be no fever and yet examination shows the lesion to be active and spreading. (vide Charts).

#### CIRCULATORY SYSTEM:

The pulse presents the chief interest. It varies with the general condition of the patient. Where the general constitutional symptoms are well marked the pulse rate is increased. The tension is low, the wave is ill sustained and the rise and fall rapid. The volume is usually small. Regularity is a constant feature; it is only towards the end that irregularity shows itself. Even in cases where the constitutional symptoms are not prominent/

prominent the rate is increased. Where the lung symptoms are slight and indefinite and associated with a frequent and low tension pulse, Pulmonary Tuberculosis should be suspected. In cases of Empysema in which there is a certain stress on the heart, an increase of the pulse rate occurs, but it is never so great nor so frequent as in Lung Tuberculosis.

Undue rapidity of the pulse is an unfavourable sign and indicates great irritability of the heart or nervous system. No doubt this is partly toxic and partly due to the fever. The pulse may be the only indication of improvement. It may not be possible to note any change in the lung condition, yet a fall of a few beats with a better tension of the pulse, warrants a more favourable prognosis.

A change in the pulse is readily produced. Slight exertion, excitement, emotion, slight haemoptysis accelerate it.

The heart, beyond its acceleration, is but little affected. It may happen that cardiac asthenia, indicated by the action being weak and the sounds, especially the first, feeble, is a prominent symptom.

Hypertrophy/

Hypertrophy does not appear to occur; on the other hand, the actual size of the heart is diminished. In 51 post mortem examinations in which the heart was weighed, it was found to average  $9\frac{3}{8}$  oz. in the male and  $8\frac{3}{4}$  oz. in the female. In 22 of the 40 males it was under the average; in 17 it was under 9 oz. In 4 of the 11 females it was under 8 oz. In a male of 47, the heart weighed 6 oz., in a female of 41 the weight of the heart was  $4\frac{1}{2}$  oz.

Wasting of the muscular system is commonly present in Pulmonary Tuberculosis, so that a certain amount of atrophy of the cardiac muscle is not surprising; yet it is in striking contrast with what occurs in other chest diseases, e.g., Bronchitis, Emphysema and Pneumonia. In these, it is often the cardiac failure that results in death, whilst in Lung Tuberculosis, death is more often the result of general asthenia.

Anaemia is often a marked feature of Phthisis, yet haemic murmurs are by no means common. The pulmonary 2nd sound is frequently accentuated and often re-duplicated.

Commonly, the heart sounds are loudly and diffusely propagated by the consolidated lung in close relation to /

relation to the heart, and with cavities near the heart, the sounds may be altered in character - ringing or metallic.

Pulsation may be seen under the cavicle in emaciated subjects and not infrequently a systolic murmur may be heard in the same situation.

The following case presented some points of interest.

J.S., 74, male, admitted with cough and difficulty of breathing. He was very deaf, so that it was impossible to get a satisfactory history of his illness.

It appeared that he had had a cough for several months, but lately it had got much worse. He was very short of breath. He had had no haemoptysis or night sweats. He had lately lost a good deal of flesh and was now easily tired out. He was at work up to 3 days before admission.

PREVIOUS HEALTH:

Six years ago, he had left sided pleurisy. His family history was satisfactory. On admission he was seen to be a well developed, but somewhat emaciated man - hair turning grey. Arcus senilis well marked. The venules on nose and cheeks were dilated/

dilated. There was no clubbing of finger ends.

His temperature was 103.6° F., pulse 96, regular, but rather poor tension, and respirations 30 per minute. He was a labourer and a heavy drinker.

#### RESPIRATORY SYSTEM:

Cough troublesom; expectoration scanty and purulent.

The right side of chest moved very little during respiration. There was well marked pulsation over whole front and in axilla on the right side. It was synchronous with apex beat and the rate of the heart could easily be counted by observing it.

There was general impairment of resonance over whole of right lung, front, back and in axilla and slight impairment of left apex. The vocal fremitus and resonance were diminished on the right side.

Auscultation of Right lung revealed vesicular breathing with prolonged expiration generally faint, but inaudible over upper part of lung. The striking feature on auscultating, was the presence of a soft systolic murmur heard over outer part of front below <sup>a</sup>calvicle, over upper part of axilla and over the whole of the back. It was loudest in the axilla over left apex; the breathing was harsh with prolonged/



prolonged and blowing expiration and a few sharp crepitations. Vocal resonance was increased. The temperature was raised during the course of the illness. The physical signs never changed. The patient got weaker and weaker and died 5 weeks after admission. (vide chart.)

The points of interest were:-

1. Pulsation over right front.
2. Systolic bruit loudest in upper part of axilla and outer part of front of chest, but heard also posteriorly.

The case at first sight suggested Aortic Aneurism. The only point in favour of it was the pulsation, but this was not expansile. The temperature might readily have been associated with aneurysm as in a case quoted by Professor Gairdner in Allbutt's System, Vol. VI. (Aneurysm of Aorta.) There were no pressure signs or symptoms and it is very unlikely that a murmur associated with an aneurysm would be heard loudest in the axilla.

The case might have been a Pleurisy with effusion, or a Pulsating Emphysema; but the absence of fluid or pus on several occasions after exploring, excluded these.

Malignant Disease of the lung or pleura had to be/

be excluded. As in Aneurysm, so in Malignant Disease the occurrence of fever did not exclude Malignant Disease. Examination of the blood failed to reveal a leucocytosis, which was against Malignant Disease.

There was no evidence of any pressure symptoms such as fluid in the pleural cavity, distended veins on the chest wall or oedema, such as one might expect in Malignant Disease. The aspect of the patient did not give one the impression of Malignant Disease. The only condition that seemed likely was Pulmonary Tuberculosis.

The history of night sweats, the temperature, the appearance of the patient and the physical signs in the other lung warranted the diagnosis. The discovery of Tubercle Bacilli later, clinched the diagnosis.

POST MORTEM:

Right Lung: Pleura very much thickened and adherent. In the upper lobe, large cavity with irregular fibrous walls. The middle and lower lobes completely consolidated.

Left Lung: No evidence of old pleurisy. Scattered tuberculous deposits throughout the whole lung. Heart weighed  $13\frac{1}{2}$  oz., very much dilated.  
The/

The aortic valves were slightly atheromatous. The pulsation was no doubt transmitted from the heart by the solid lung.

The systolic murmur could have been vascular in origin and due to pinching of a vessel by a fibrotic portion of lung, or to pressure by glands. There was, however, no evidence of either.

More probably, it was pulmonary in origin and caused by air being forced out of the cavity during the systole of the heart.

#### ALIMENTARY SYSTEM:

From an analysis of the 80 cases under review, I find three of them commenced with symptoms pointing to a disturbance of the Alimentary tract, such as loss of appetite, vomiting or diarrhoea.

Digestive disturbance is no necessary part of Phthisis, but it stands in close relation to the constitutional signs, being as a rule considerable where these are well marked.

Cases are met with in which all the symptoms point to the Alimentary Canal. These cases are most frequent in young women and unless a careful physical examination of the lungs be made, an error of diagnosis will result. Such cases are frequently/

ly looked on as cases of Anaemia, Dyspepsia, or Gastric Ulcer.

The appetite may be good throughout, more frequently it is capricious. A good appetite is a hopeful sign, but a loss of appetite and refusal of food is of bad omen. When the digestion fails, the ordinary symptoms of dyspepsia present themselves - flatulence, nausea, vomiting, pain after food, colic, irregularity of the bowels and diarrhoea.

#### VOMITING:

Is by no means uncommon; 28% of my cases gave a history of it, the female sex showing it more frequently than the male in the proportion of 3 to 2. Whilst under observation in hospital, vomiting was much less frequent and no special drug treatment was carried out.

Loss of appetite rarely occurred. The infrequency of both vomiting and loss of appetite, I think, can be accounted for by treating the patients on open-air principles.

The vomiting, when it did occur, was more often mechanical as the result of coughing, rather than dyspeptic.

Two cases presented well marked ulceration of the tongue. This complication causes the patient much discomfort and pain, and like Tuberculous Laryngitis, is a hindrance to the taking of food. Difficulty or pain on swallowing depends on ulceration of tongue, pharynx or larynx. It may also be caused by pressure of enlarged glands on the oesophagus in the mediastinum. I had one such case.

The patient was an old man, 62 years of age. He had had Chronic Pulmonary Tuberculosis for three years. For 12 months before death, he complained of difficulty of swallowing, which increased so much that he could only swallow liquid food. There was occasionally slight dyspnoea and pain, referred to middle of sternum. Post mortem examination revealed enlarged caseous glands pressing on the oesophagus.

#### DIARRHOEA:

Occurred in 38.6% of the cases and wastwice as frequent in the female sex.

Diarrhoea may occur independently of amyloid disease or ulceration.

None/

None of my cases showed any evidence of Amyloid and in those cases in which post mortem examinations were made, ulceration was almost always found, but diarrhoea stands in no necessary relation to ulceration, for the latter may be widespread and yet there may be no diarrhoea.

When ulceration is present, diarrhoea may be readily excited by errors in diet or by drugs.

The influence of drugs is well shown in the case of a woman, 35 years of age, admitted complaining of diarrhoea of three days' duration. Her history was that three days previous to admission, she had taken  $\frac{1}{2}$  oz. of Epsom Salts and since then, she had had severe diarrhoea. For the previous six months, she was troubled with cough, night sweats and obstinate constipation.

On admission, she was seen to be in a collapsed condition, was very emaciated and looked extremely ill. She died 24 hours after admission; post mortem examination revealed infiltration of both upper lobes and widespread congestion and ulceration of the greater part of small intestine and the whole of large intestine.

Haemorrhage/

Haemorrhage from the bowel occurred in one case. This was a man, 64 years of age, with infiltration of right apex and Right Tuberculous Pleurisy. At no time was there any diarrhoea. The bleeding was profuse, 20-25 oz., and was not repeated.

Post mortem examination showed involvement of right apex and a general pleurisy on same side of a very haemorrhagic character. Numerous ulcers were found throughout the greater part of the bowel.

Ischio-rectal Abscess or Fistula did not occur in any of my cases. These appear to be rare complications. Wilson Fox's figures yield a percentage considerably under one.

THE RELATION OF ALCOHOLISM AND CIRRHOSIS OF THE  
LIVER TO PULMONARY TUBERCULOSIS.

Alcoholism and Phthisis are both such common affections that they must of necessity be not infrequently associated by way of accident, but any closer relation between them is difficult to prove.

Whether or not phthisis be more common amongst alcoholics, I think it runs a more rapid course in an alcoholic.

At one time it was suggested to treat Pulmonary Tuberculosis with large doses of Alcohol in the belief that it would produce in the lung cirrhotic changes like those produced in the liver. This has now been shown to be an error; the fibrous changes when they occur, are of tubercular origin. " In any series of alcoholic cases, a good number of them die of Phthisis (West). Leudet's figures show a percentage of 12.5 and in extreme cases as high as 17.

Of the total number of cases for the past year admitted into this hospital, 14% of them were cases of Pulmonary Tuberculosis, and the majority of these were confirmed drunkards. According to West, in any series of cases, Cirrhosis of the Liver is not common./

common. His own post mortem figures yield a percentage of 1.5, Leudit's percentage is higher, viz., 3. In my series of cases, 4 had associated Cirrhosis giving a percentage of 5.

All of them occurred in males. Out of a total of 1152 admissions for the past year, 6 showed Cirrhosis of the Liver and of these, 4 had Pulmonary Tuberculosis.

Thus, the infrequency of Cirrhosis and the frequent association of Tuberculosis of the lung with it is striking.

The majority of those admitted to Workhouse Hospitals being heavy drinkers, one would expect Cirrhosis to be more frequent, yet the above figures show it to be very rare. Of the 4 cases, 3 of them presented the typical hobnailed character of alcoholic cirrhosis, the remaining one showed a very shrunken liver, the left lobe being represented by 1 oz. of liver tissue and was quite separate from the right lobe and was ductless. The liver did not feel particularly hard; the surface was mammillated and the capsule thickened. The liver had not the firm feel or cut one associates with the true cirrhotic liver. The patient was a fairly temperate man. The liver in these cases weighed:- 46½ oz. - 42 oz. - 40 oz.

In from 70 to 80% of the cases of Alcoholic Cirrhosis, a history of alcoholic excess is obtained. This alcoholic excess is widespread, yet Cirrhosis cannot be said to be so common nor so widespread.

It is a common enough disease in London; it is uncommon in Scotland (Hal~~l~~ White.)

Then again, cirrhosis of the Liver is sometimes found in children and in them, alcohol as the cause is out of the question.

These facts suggest that alcohol per se is not the cause of Cirrhosis. Then again, the epidemic of Peripheral Neuritis in and around Manchester opens our mind in the same direction. The cause was believed to be alcohol, until Arsenic was discovered. So perhaps also in Cirrhosis, it is not the alcohol. The alcohol may act as a predisposing factor. It may be that the exciting cause is some deleterious agent in the alcohol. The combination of Pulmonary Tuberculosis and Cirrhosis is so frequent that one is tempted to look on them as cause and effect.

Alcohol may merely predispose to Cirrhosis and various toxic agents may excite it. Possibly the products of Pulmonary Tuberculosis may have this role.

PULMONARY TUBERCULOSIS AND DIABETES MELLITUS.

It is rare for Diabetes Mellitus to develop in the course of Pulmonary Tuberculosis; on the other hand, a large proportion of cases of Diabetes Mellitus die of Phthisis.

One of my cases of Pulmonary Tuberculosis developed in a man with Diabetes Mellitus.

He was a waiter, 28 years of age, who was admitted on the 26th November, 1901 complaining of thirst, polyuria and general weakness. He had been a heavy drinker of spirits and lived in lodgings. On admission, he was seen to be a spare man with puffy pale face. His skin was dry and harsh, and his breath had a sweetish odour. His appetite was not voracious. Nothing abnormal detected in the abdomen; his bowels were constipated. The circulatory and respiratory systems were healthy. His pulse was 50 per minute, regular and of moderate tension. The fundus was normal. His knee jerks were absent and he complained of cramping pains in the calf muscles. He was placed on ordinary diet. During the first week after admission, he excreted on/

on an average 170 ounces of urine and 1530 grains of glucose. He was then put on diabetic diet. On this diet, the average excretion between 24th July, 1902 and 8th February, 1902 was 156 ounces of urine and 825 grains of glucose. One grain of Codeine afterwards increased to one and a half grains was then administered twice daily. The amount of urine and glucose excreted fell immediately and remained fairly stationary, the average output being 74 ounces of urine and 254 grains of glucose in the day.

His weight so far, remained fairly stationary; on the 13th of August, he weighed 8 st. 9 lbs. His pulse had increased in frequency, varying between 80 and 90 per minute. A few days later, he complained of cough. His temperature, so far sub-normal, was now slightly elevated - 99° F. Examination of the chest revealed an infiltration of the apex of the Right lung. Tubercle bacilli were found in the ~~urine~~. *Sputum*.

On the 30th August, he was put on 4 oz. of ordinary bread and on the 19th September, the diet was changed to ordinary diet, but the codeine was continued as before. The temperature was now oscillating between 97° F. in the morning and 103° F. in the evening and the pulse was correspondingly increased in frequency. The lung condition was found

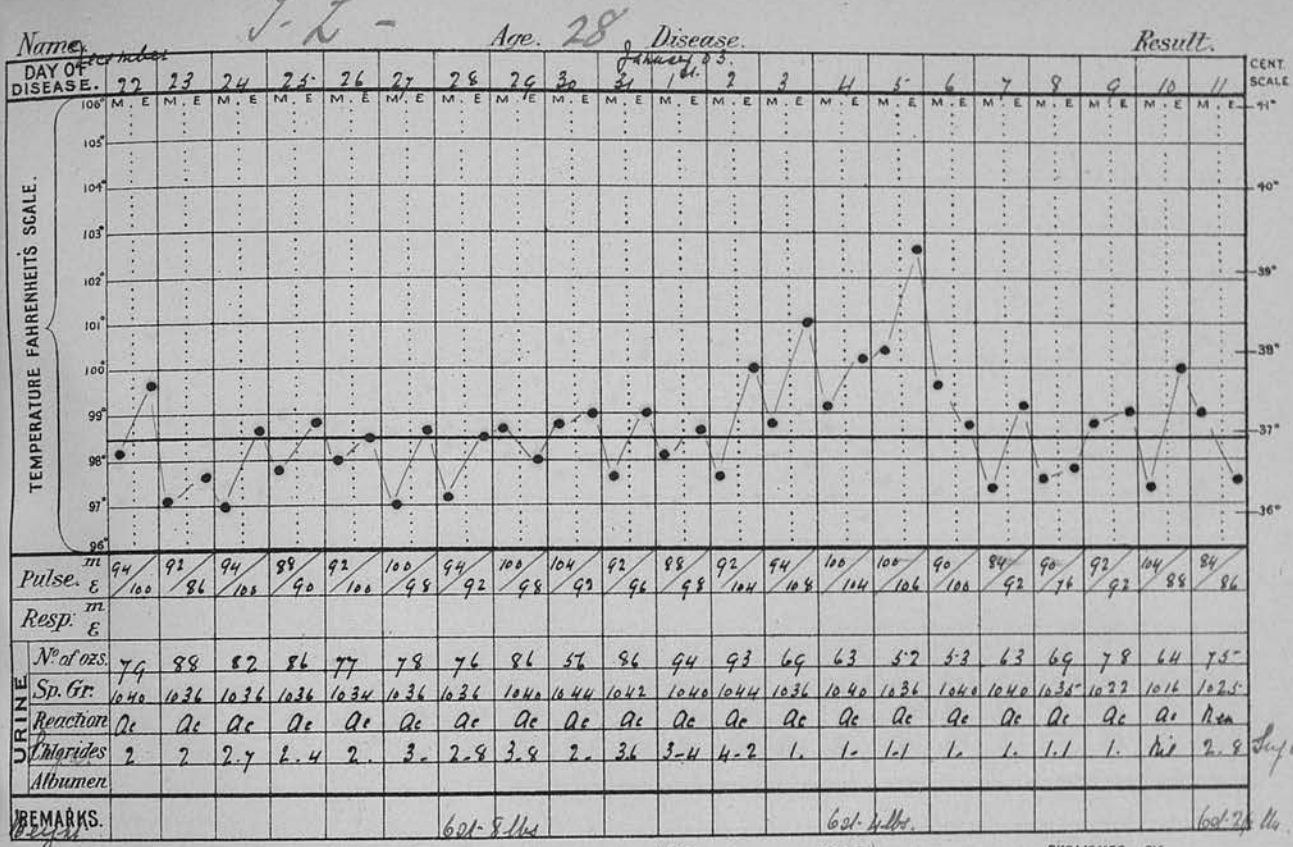
to be rapidly spreading. Between the 20th of August and the 19th of September, the average output was 72 ounces of urine and 357 grains of glucose. He was now steadily losing weight and felt much weaker. The cough was troublesome and expectoration profuse and purulent. From 19th September to 18th October, he excreted on the average, 75 oz. urine and 395 grains of glucose. Between these dates, he was having 20 grains of urea thrice daily.

The whole of the right lung was now consolidated and the left upper lobe infiltrated. In the first week of June, 1903, his weight was 7 st. 4 lbs. The temperature had quietened down somewhat, but between the 1st and 9th January, it showed greater elevations. Between these dates, the average output was 71 ounces of urine and 131 grains of glucose. Several days later, the sugar disappeared from the urine. The Codeine was then stopped. The cough continued troublesome and the expectoration profuse. He perspired freely during sleep. On the 4th February, the lesion was found to be very active and spreading rapidly in the left lung. The right side of the heart was dilated and the lower extremities oedematous. His weight had fallen to





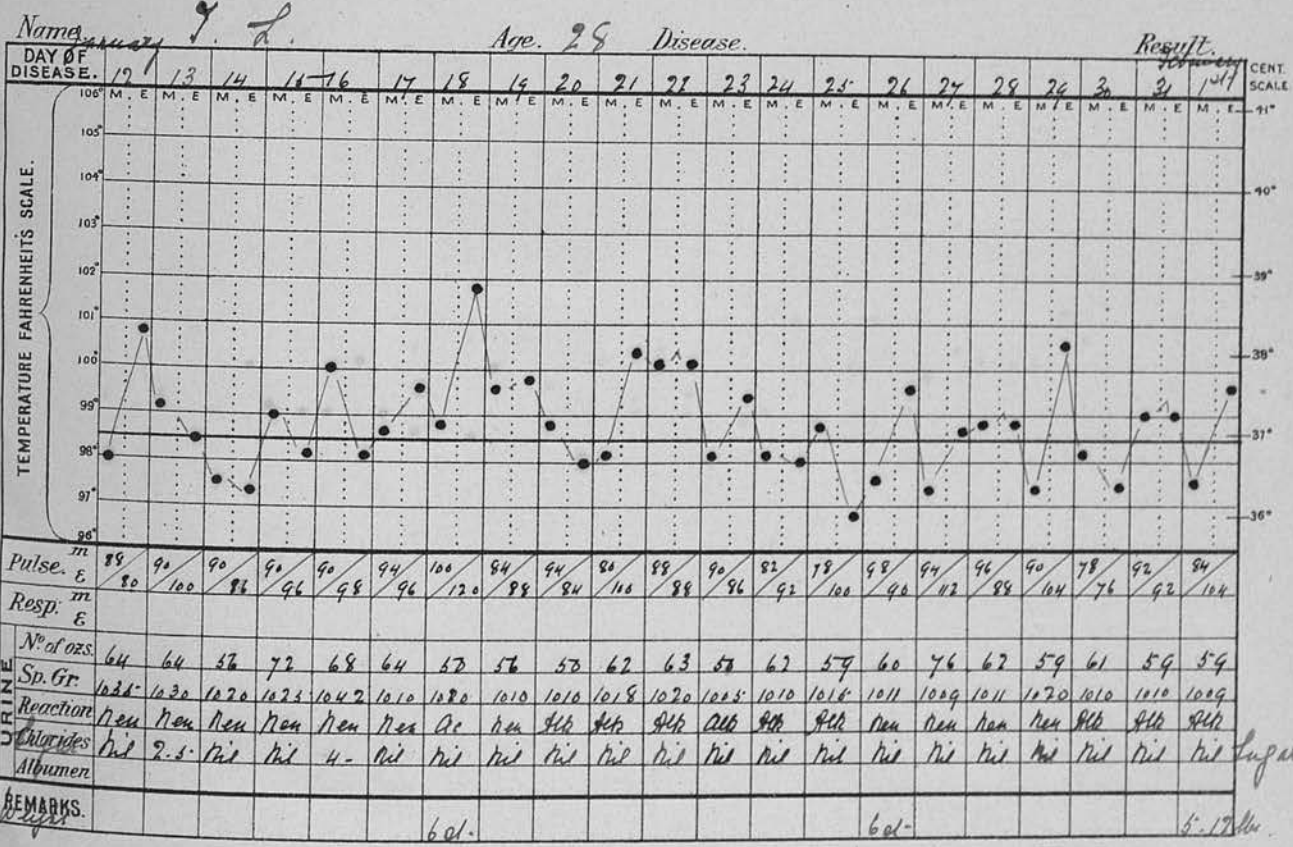
CLINICAL CHART OF TEMPERATURE & C.



(Observations taken at ..... A.M. and ..... P.M.)  
For Memoranda of Treatment, see back of Chart.

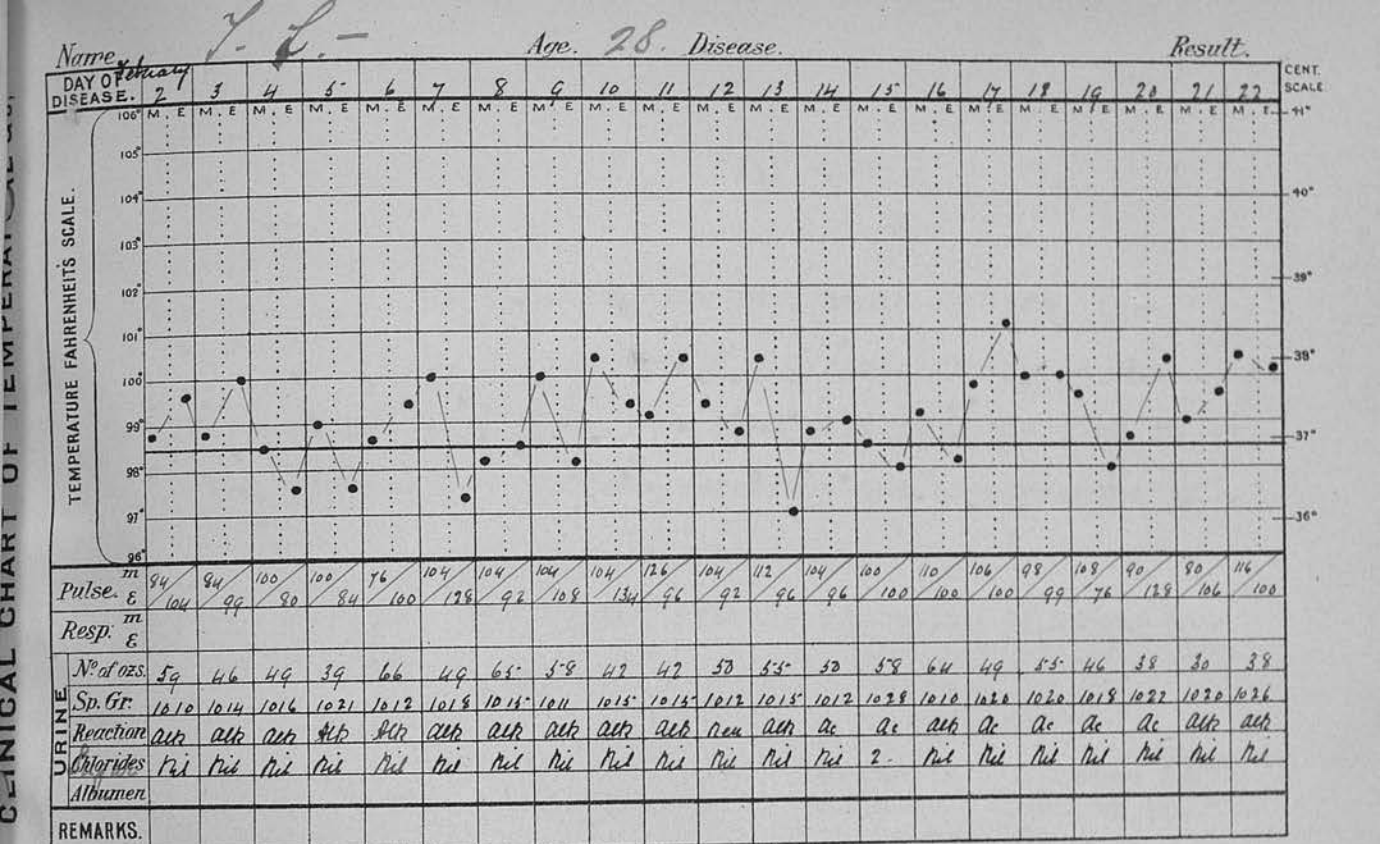
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CLINICAL CHART OF TEMPERATURE & C.



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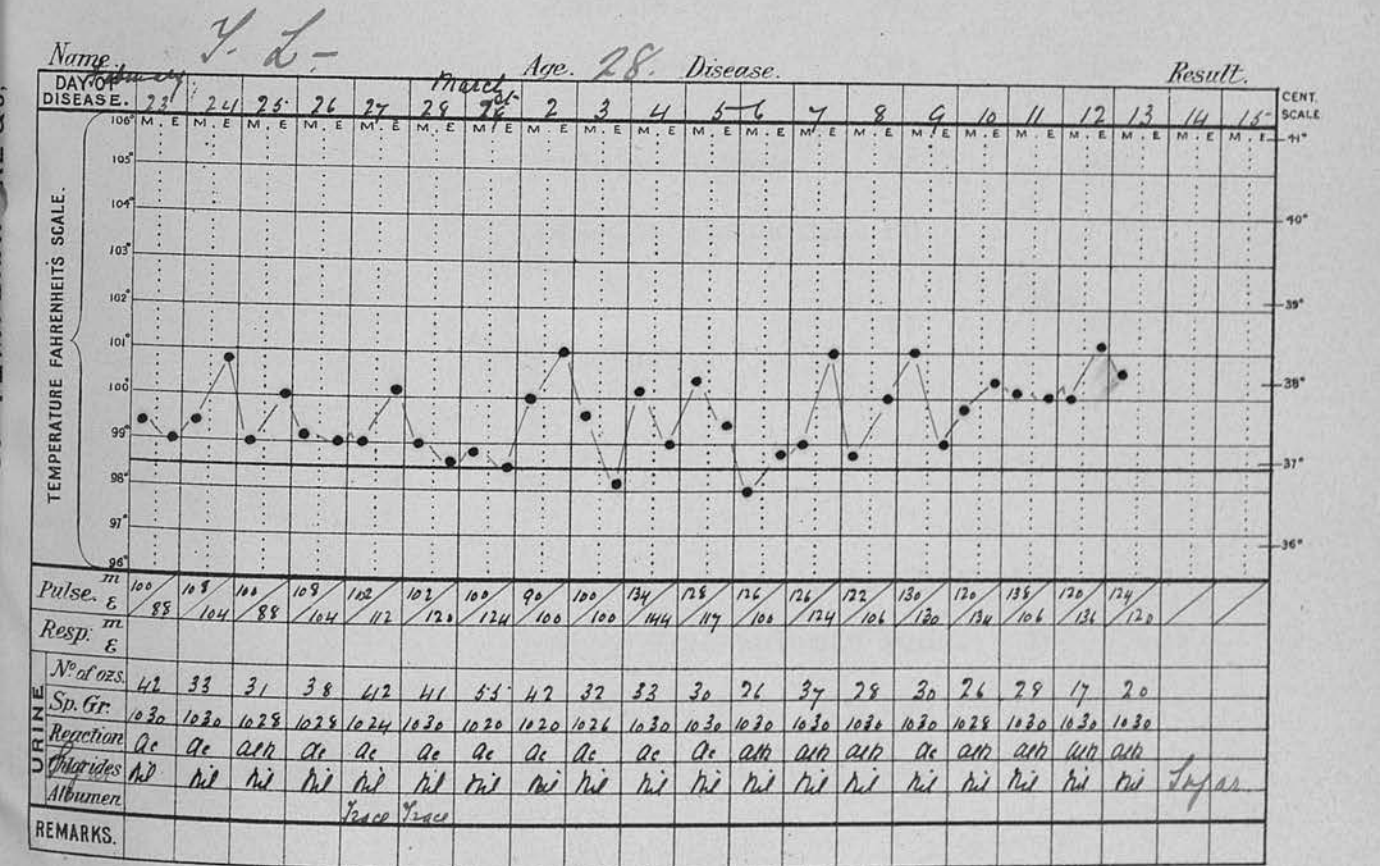


(Observations taken at AM and PM)  
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156

825



(Observations taken at AM and PM)  
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6 st. 8 lbs. During the last month of life, the temperature was constantly elevated, rising in the evenings to 101° F. He gradually sank and died on 15th March, 1903, two years from the commencement of his illness.

Table showing the average excretion of urine and glucose in 24 hours.

		Urine in ounces in 24 hrs.	Glucose in grains in 24 hrs.
1.	On admission, ordinary diet	170	1630
2.	Diabetic Diet,	156	825
3.	Diabetic Diet Codeine,	74	254
4.	Diabetic Diet 4 oz. Bread Codeine,	72	357
5.	Ordinary Diet Codeine Urea	75	395
6.	Ordinary Diet Codeine (Subnormal Temperature),	88	217
7.	Do., (Rise of Temperature.)	71	130

REMARKS:

The case shows the rapid course Diabetes complicated by Pulmonary Tuberculosis runs. For close on 18 months, the patient held his own fairly well; after the development of the lung mischief 6 months before/

before death, he steadily went down hill. The diabetes was not of a severe type. The lung lesion was active throughout and spread rapidly. It was of the caseating pneumonic type. When the lung condition developed, the diet was changed to ordinary diet. The table shows a very slight increase in the amount of sugar and urine excreted. According to Dr R. T. Williamson, the sugar output is diminished when phthisis complicates Diabetes Mellitus. Very probably, in this case, if the Diabetic Diet had been persisted in, the amount of sugar excreted would also have been less.

A temporary rise of temperature with a coincident spread of the lung lesion appears to be followed by a diminution of the urine and glucose output (vide Table.)

The sugar disappeared from the urine two months before the end. During the pre-tubercular period, the weight remained fairly stationary, but with the development of phthisis, the patient lost weight rapidly (vide temperature charts.)

URINARY SYSTEM:

There are no characteristic changes in the urine in Pulmonary Tuberculosis. The urine, in my experience, is remarkably free from the presence of albumen. In febrile conditions the urine presents the usual characters and transient albuminuria may occur, but this, I think, is less frequent than in other febrile conditions. Cases are seen in which the temperature is persistently raised for weeks and yet there is no albumen in the urine.

In chronic cases, albumen is more frequently present, and in some of those it is associated with oedema. A proportion of such cases is due to cardiac weakness. With rest and improved health both oedema and albuminuria disappear.

In the remaining cases with albuminuria, it is generally the result of amyloid disease or of granular kidney. Nineteen per cent. of my cases showed persistent albuminuria. Wilson Fox found albuminuria in 8% of the acute cases and 33% of the chronic, giving a total of 41%. The difference is considerable, but Fox's figures may include cases with transient albuminuria. If we are to look on Pulmonary Tuberculosis as a general toxæmic state, then we might expect albuminuria to be more frequent, and to bring it in line with other toxic states, e.g., Scarlet/

Scarlet Fever, Diphtheria, etc.

Many of the chronic cases lasting for years may show no evidence at any time of albuminuria. Absence of albumin in the urine may be due to the toxic products, either not being excreted by the kidneys, or because of them not having a deleterious action on the kidney parenchyma. Possibly the toxic products are excreted by other channels, such as the skin glands - the troublesome sweating of Phthisis may be the result of stimulation of the secretory nerves or sweat glands by these products.

West states the excretion of urea to be about normal, i.e.,  $1\frac{1}{2}$  to 2%. The difficulties and errors that may arise in estimating the excretory output of urea in the urine are manifold. The daily output may vary very much, and so give rise to false conclusions if a few isolated observations be made. This source of error I have excluded by making daily estimations. Another difficulty which arises, introducing a source of error, is that of impressing upon the patient the necessity of saving every drop of his urine. The conclusions arrived at, however, are based upon the results taken as a whole. The apparatus used was Southey's ureometer.

Dr/

Dr H. Harper has forcibly advocated the opinion that urea exerts a specific action on Tuberculosis (Lancet, March 9, June 15, December 7, 1901.) I have submitted this treatment to a prolonged trial, and I estimated the urea excretion before and during the administration of pure synthetised Urea. All the cases were of a chronic type, but they varied in age, build and temperament and the disease was not in all at the same stage.

#### CASE I.

J.H., 52, single, labourer. He had been troubled with cough in winter for the past 16 years. For 7 months previous to admission, he had had a troublesome cough with profuse purulent expectoration. Never had haemoptysis. No night sweats, no vomiting, no diarrhoea. His chief complaint was loss of flesh and weakness. He was very anaemic and emaciated and 9 months previous to admission, he weighed 10 stones. When admitted to hospital, his weight was 6 st.  $1\frac{1}{2}$  lbs. He was a heavy drinker and had not worked for 5 months previous to admission.

The physical signs were those in infiltration and/

and consolidation of whole of left lung and upper lobe of right. A few sharp creps were heard at right apex and left base. His temperature varied from 97° to 99°F., and pulse 72-90 was regular and rather poor in quality.

Two months after admission, the physical signs had improved; there were fewer accompaniments, and expectoration less profuse. Patient's general condition was the same as on day of admission. His weight was now 6 stones, and the pulse and temperature unaltered.

He was then put on 20 grs. Urea thrice daily. He took in all 6 oz. of Urea. Not the slightest improvement could be noted during, or after the administration of the Urea. The appetite did not improve and he steadily lost weight. The pulse rate increased slightly in frequency. The lung condition became more active, the lesion having spread a little. The temperature remained unchanged. The patient weighed 5 st. 7½ lbs. when he was discharged from hospital.

#### CASE II.

P.B., 59, labourer, admitted on 29th July, 1902, complaining of cough of 7 months duration. Previous/

Previous to this he enjoyed good health and never had any important illness. He had been a heavy drinker. There was no history of haemoptysis. When in health, his weight was 12 st. 2 lbs. On admission, he weighed 9 st. 2 lbs. In the chest there were signs of infiltration of right upper lobe. His pulse varied from 72 to 100 and was of low tension. The temperature was 97° F. in the mornings and usually 100° F. in the evening. On 27th September, examination of chest revealed an infiltration of left apex. Pulse rate had now increased, being between 90 and 100, but temperature had quietened down. Cough was troublesome, but the expectoration was scanty. He had gained 5 lbs. in weight and on the whole, his general condition was better.

In February, 1903, examination showed both upper lobes were consolidated. Weight 9 st. 6 lbs. and pulse now varied from 70 to 84 and of better tension. The lung condition appeared to have quietened down.

On March 1st, 1903, he was put on 20 grains of Urea thrice daily, which was gradually increased. He took in all 10 oz.

No improvement could be noted. The cough and amount/

amount of expectoration remained the same. There was no improvement in his general condition and he had now lost 4 lbs. in weight. His weight was now the same as on day of admission, viz., 9 st. 2 lbs.

### CASE III.

H.G., 41, tailor, admitted 4th August, 1902. The duration of his illness was 18 months and began with cough. He was expectorating a large amount of purulent sputum, which from time to time had been blood streaked. He had had no vomiting and no diarrhoea. He had done no work for the previous 12 months. His family history was satisfactory.

On admission, he had signs of consolidation of lower lobe of left lung. The temperature was seldom above 98° F. His pulse was regular, rather increased in frequency and of low tension. His appetite was not good. His weight on admission was 7 st. 11½ lbs. For several months he held his own, gaining a few pounds in weight and feeling more comfortable.

In December, the lesion of the left lung had quietened, but an area of infiltration was noted in the right upper lobe. The pulse had rather increased in frequency. In March, he was put on 20 grains thrice daily. He took in all, 6 oz. If anything/

anything, the patient had improved in his general condition, his weight being 7 st.  $13\frac{3}{4}$  lbs. Expectoration was quite as abundant. No change in the pulse or temperature could be noted.

#### CASE IV.

The patient was a married man, 27 years of age and had been through the South African Campaign. Symptoms had been present for 2 years. During this time he had had cough with scanty expectoration. There had never been haemoptysis.

His previous health had been good, and his family history was satisfactory. On admission, the signs pointed to infiltration of both upper lobes. His weight on admission was 8 st. The pulse was 70 per minute, regular and of low tension.

One month after admission, he had improved somewhat. There were fewer accompaniments in the lungs and appetite was good. Notwithstanding he had lost 2 lbs. in weight. He was put on 20 grs. of Urea thrice daily, the dose being gradually increased. He took 12 oz. of urea.

No improvement could be noted. The physical signs in the chest were unaltered. The pulse had quickened/

quicken, varying from 80 to 104 per minute. The temperature remained as before, subnormal. There was no gain in weight. When the urea was stopped, he weighed 7 st. 12 lbs. During urea administration, he complained of headaches and feeling out of sorts.

REMARKS:

The lesion in these cases was well marked and of a chronic type. Two of the patients gained a few pounds in weight during the urea period, but the gain was no more than that seen in other patients not taking urea.

One lost weight and the other remained stationary. Urea appeared in no way to improve the appetite of the patients. No appreciable improvement could be noted in the physical signs. Urea appeared to exert no special influence upon either the pulse or temperature, and in no case was there any marked diminution in the quantity or any appreciable improvement in the quality of the expectoration. The amount of Urea given was not so much as that administered by Dr Harper.

In an earlier series of cases, I gave it continuously/

tinuously for months as much as 20-30 ounces being administered. In these earlier cases, as in the present series, the conclusion come to was that Urea in no way exerted a beneficial influence on cases of Pulmonary Tuberculosis.

Before relating the conclusions, I will give briefly the main outline of the results of my investigations.

TABLE I.

Showing the average output of Urea in grains per 24 hours.

Table I. Output of Urea in grains per 24 hrs.

Case	During Non-urea period	During urea period	During 1st fortnight of urea period	During last fortnight of urea period.	Length in days of Urea period
A.	376	389	404	362	55
1	128	297	227	406	41
2	342	450	589	389	59
3	269	332	342	323	46
4	420	523	564	559	64

Case A. shows the excretion of urea by a healthy young man, 17 years of age.

The table shows:-

1./

1. That in Pulmonary Tuberculosis the excretion of Urea is diminished.
2. That during the administration of urea the output of Urea is increased.
3. That this increase is greatest at the beginning, but tends to diminish.

Urea given by the mouth is recommended by Strumpell as a diuretic. This point I have investigated. The accompanying table shows that Urea has a slight diuretic effect; that this is more marked at first, and tends gradually to pass off.

TABLE II.

Showing the daily quantity of urine during the non-urea and urea periods and also the average quantity during the early part of the administration of urea and during the later part.

Case	Average daily quantity of urine during non-urea period.	Average daily quantity of urine during urea period.	Average during the first 14 days of urea period.	Average during the last 14 days of urea period.
A.	52	55	60	54
1	37	42	48	39
2	56	61	62	61
3	42	49	50	48
4	63	78	83	70

The/

The therapeutic action of Urea may be summarised as follows:--

1. Urea given by the month in patients with Chronic Pulmonary Tuberculosis increases the output of urea.
2. Urea acts only to a slight extent as a diuretic.
3. In both, the output of urea and in the output of urine, the effect produced by urea is most marked at first. The body seems to accustom itself to the intake of urea and tends to return to the normal output.
4. Urea has no action as a cardiac stimulant.

RELATION OF PHTHISIS TO DISEASES OF THE NERVOUS SYSTEM

According to West, a good many cases of Alcoholic Peripheral Neuritis die of Phthisis.

In Diabetes Mellitus, Phthisis is a frequent complication and in it, as an Chronic Alcoholism, peripheral neuritis occurs.

It has been suggested (Allchin's Manual of Medicine) and indeed seems probable that the lung condition in Alcoholism and Diabetes is favoured by the occurrence of Neuritis affecting the Vagus Nerve.

This vagal neuritis may:-

1. Act by lowering the tone of the pulmonary vessels, producing a paralytic condition leading to congestion and oedema of the lungs.
2. Take the form of a Tropho-neurosis.

Alcoholism and Diabetes are general diseases with a toxic factor in both producing pathological changes, e.g., Neuritis <sup>and</sup> Cirrhosis.

In like manner, Pulmonary Tubercle may very well be looked on as a constitutional disease in which there is a toxic factor acting on the various systems./

systems. Thus we may have the correct explanation of the rapid heart, the sleep sweats, the wasting, the peripheral neuritis, the peculiar mental attitude and the insanity of Phthisis.

Peripheral Neuritis occurs as a complication of Lung Tuberculosis. This tends to bring the latter into relation with other germ or specific fevers such as Diphtheria, Typhoid or Malaria, with all of which, peripheral neuritis may be associated. In the following case, Peripheral Neuritis complicated Pulmonary Tuberculosis.

G.K., 53, Hawker, admitted complaining of Cough and weakness. Seven months ago he developed a cough. He never had haemoptysis. Occasionally sweated during the night. He had steadily been losing weight. For a week before admission, his legs felt very weak. He had not been confined to bed and on the day of admission was hawking. Previous health: Gonorrhoea thirty years ago. No other illness of any importance. His family history was satisfactory. He was unmarried and lived in a common lodging house. He was a non-smoker, but had been a heavy drinker until seven months ago; since then, he had been teetotal.

Formerly/

Formerly was a watchmaker, but for past five months been hawking. On admission he was seen to be a fairly well developed man, somewhat emaciated; his muscularity was fair. His voice was hoarse.

Pulse was 96, regular, but of poor tension. Temperature 99.2° F. Examination of his chest revealed a well marked consolidation of the left upper lobe. The lung condition did not appear to be active.

Examination of throat showed a small ulcer on left vocal cord. There was no albumen in the urine.

#### NERVOUS SYSTEM:

Mentally, he was somewhat dull and apathetic. Sensation was normal. Deglutition was unimpaired and the bladder and rectal functions were intact. The plantar reflexes were sluggish and the other superficial reflexes were absent. The knee and tendo Achilles reflexes were lost on both sides.

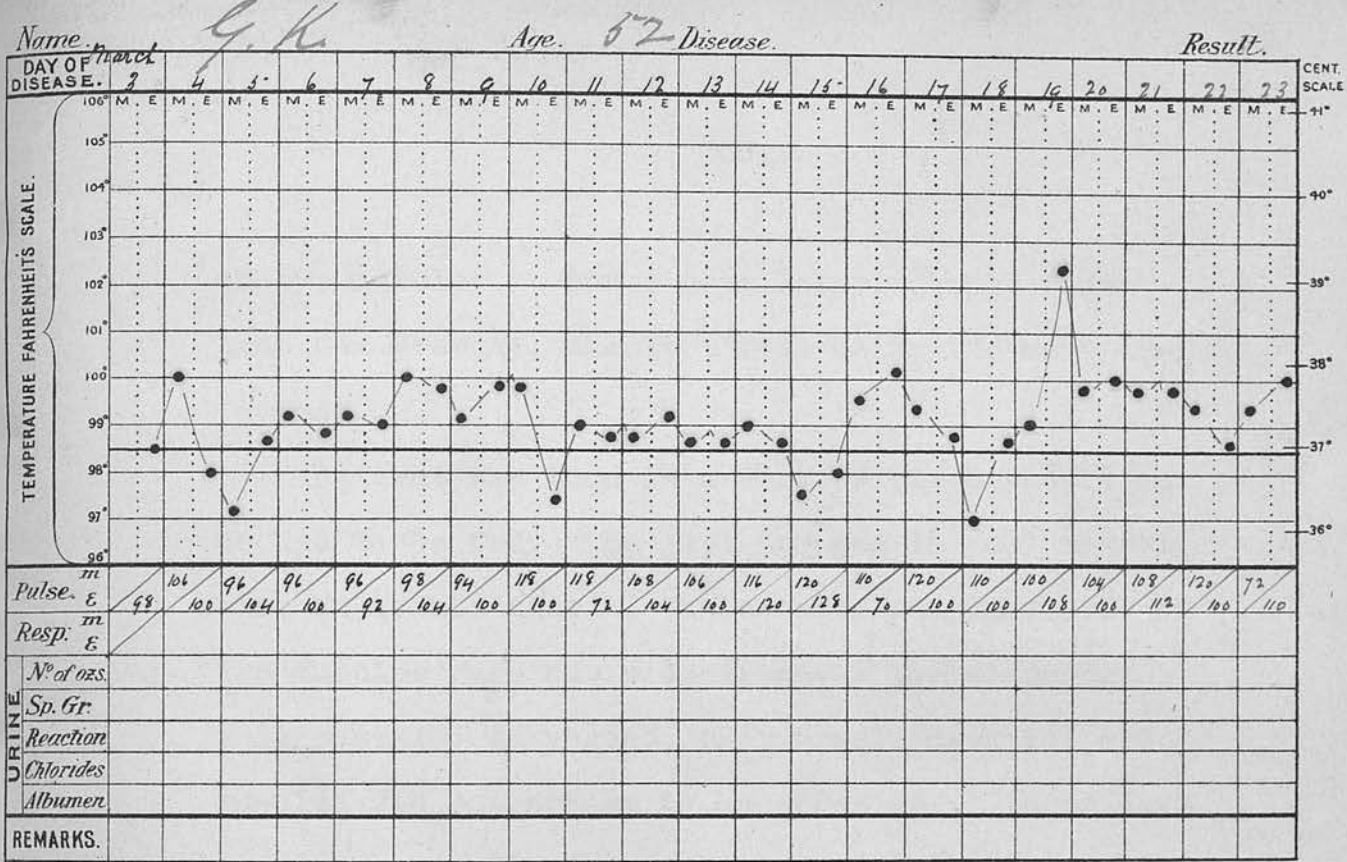
The muscles were soft and flabby and loss of power was a prominent symptom, especially in the arms and legs. Certain groups of muscles had undergone a certain amount of atrophy. This wasting was seen chiefly in the extensor group of the fore-arms/

arms and in the Tibialis Anticus and the Extensors of the toes. To a less extent the Deltoid biceps and supinator longus were atrophied. The small muscles of the hand were but little affected. The reaction of degeneration was obtained in the majority of these muscles.

Voluntary muscular power was decidedly diminished in the arms and legs, especially on the left side. The gait was atoxic with a tendency to raise the feet high. Romberg's sign was not present. The pupils were equal, moderately contracted and reacted to light and accommodation. The discs were normal. One week after admission, the following note was made.

Patient looks brighter. Appetite is good. The loss of power has increased; there is double wrist drop. Patient cannot feed himself. The trunk muscles are weak; patient gets out of bed with difficulty. He is unable to walk. There is slight disturbance of sensation in body, arms and legs. The muscles of trunk and other muscles of limbs show a certain amount of atrophy. Reaction of Degeneration obtained.

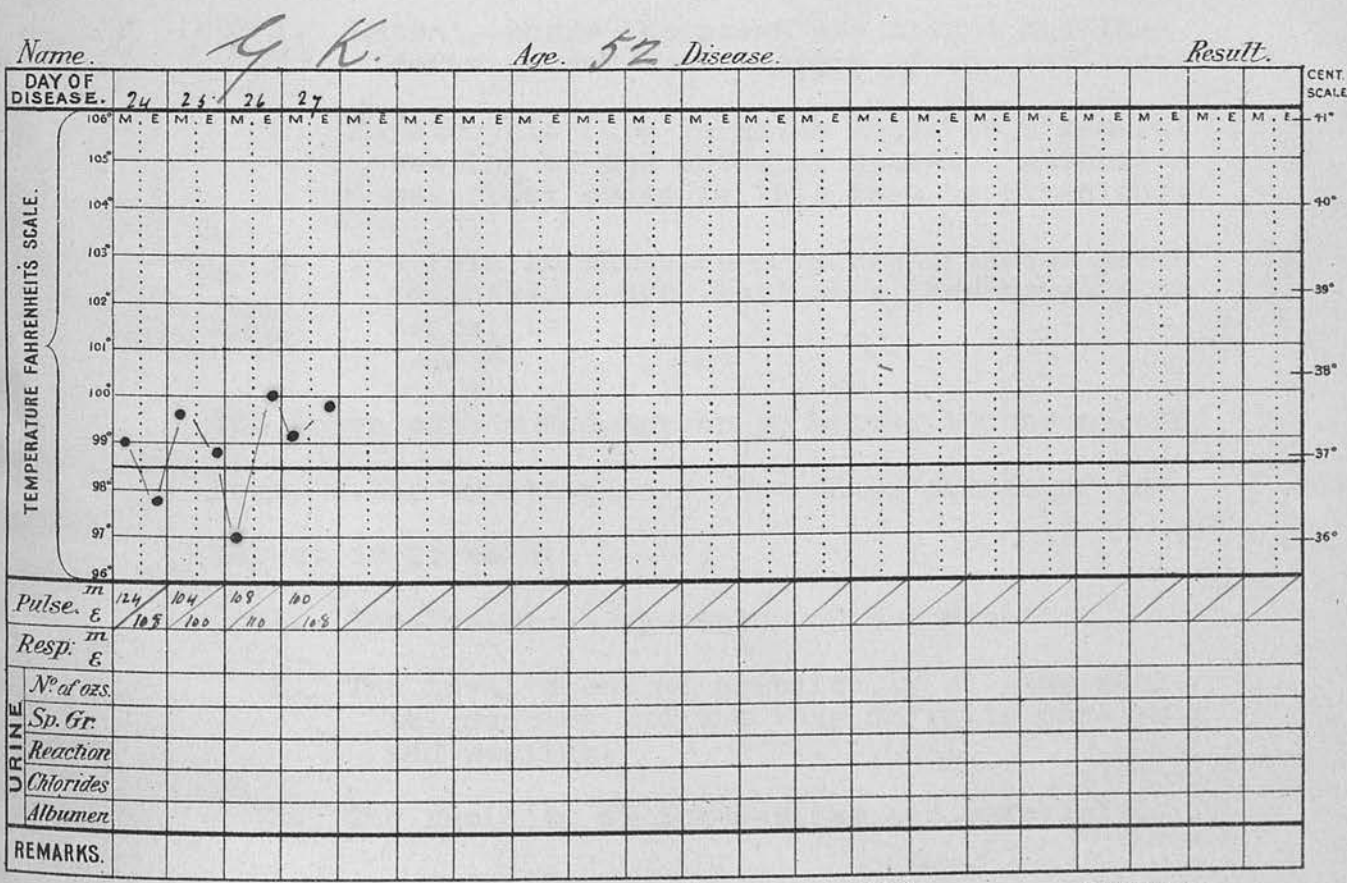
The sphincters are unaffected. No paralysis  
of/



(Observations taken at ..... AM and ..... P.M.)  
 For Memoranda of Treatment, see back of Chart.

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forms (West)



(Observations taken at ..... AM and ..... P.M.)  
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of eye muscles. Three days later, double foot drop was present, the left foot being the more affected.

Patient was then in a very helpless condition. He had to be fed. He just managed to roll himself over on to his side. The atrophy and paralysis of the muscles were now widepread. The diaphragm intercostals, laryngeal, pharyngeal, neck and eye muscles did not appear to be affected. Three days later, he was found dead in bed. (vide Chart.)

Peripheral Neuritis in Phthisis occurs in three forms (West).

1. Latent, where the pains are slight and indefinite and their nature often overlooked.
2. Amyotrophic form in which there is a general wasting of the muscular system. Attention was first drawn to this form by Eisenlohr.
3. The form in which sensory disturbances predominate, - hyperaesthesia, anaesthesia or neuralgia.

The above case would appear to belong to the second form, i.e., amyotrophic. The chief points of interest in it were:-

1. The insidious character of the onset.
2. The involvement of practically all the skeletal muscles and the very definite paralysis and wasting.
3. The rapidity of the wasting and paralysis.
- 4./

4. The mode of termination; the patient died suddenly. Death was probably due to involvement of the muscles of respiration.
5. The lung condition was of the chronic type and was in a quiescent state.

Pulmonary Tuberculosis is common in the insane; according to Clouston, it is four times as frequent. Two thirds of all the idiots die of Tuberculosis of the Lung. There can hardly be any connection between mental disease and phthisis other than that the general health is greatly impaired, so that little resistance is offered to infection.

Phthisical persons may become insane. According to Clouston, phthisical insanity forms 3 per cent. of all the cases of insanity.

Of the 80 cases under consideration, three of them terminated in insanity.

#### CASE I.

S.B., 42, by occupation a bookmaker, admitted on the 27th March, 1902, complaining of pain in the left side and cough.

He was a poorly developed man, markedly anaemic, hair scanty and skin dry. Forehead receding; finger ends were clubbed.

There/

There was no history of haemoptysis. He sweated frequently at nights. On admission, he weighed 7 st.  $4\frac{1}{4}$  lbs. His previous health was good. His family history showed that his mother was subject to fits, and one brother drowned himself. His father, seven brothers and two sisters were alive and well. He was an unmarried man and a heavy drinker and had undergone several long periods of imprisonment for theft.

Examination of chest revealed infiltration of left upper lobe. For the first week his temperature was raised, ranging between  $99^{\circ}$  and  $102.4^{\circ}$  F.; it then quietened down, being of a "sub-febrile" type with occasional elevations.

The pulse rate was increased at time of admission and rather increased in frequency as time went on. It was regular but of very low tension. His appetite was fair. There was no albumen in the urine. He slept well.

Fourteen days after admission, he became emotional, crying and laughing for no apparent cause. Sleep was disturbed; he was very restless and talkative at nights. He was good humoured, jocose and talked recklessly. The lung condition had/

had now spread, the whole of left lung being in a state of consolidation. The temperature was raised and pulse rate increased. His mental condition steadily got worse.

At times he became very excited, constantly in and out of bed. He talked incessantly, garrulous and at times was very obscene. At times, showed great vanity of dress, frequently dressing and undressing himself. He was very suspicious and showed great dislike to some of the patients and nurses. He accosted every one in flippant terms. Frequently picked the patients' lockers when they were asleep. He became more and more emotional; gesticulating and grimacing when he knew he was being watched. When spoken to sharply, he became rational and obedient, but soon lapsed into his old condition.

He steadily lost weight and his complexion was not earthy. There was no defect of articulation. The pupils were equal and reacted to light and accommodation. He ultimately became unmanageable and had to be transferred to the Asylum six months after admission to Hospital. He died three months later. The lung lesion was of a subacute character and of not/

not more than two years' duration. The mental symptoms lasted nine months. The chest symptoms were not prominent. The appetite remained good, but he lost weight somewhat rapidly. The mental state appeared to be that of Defective inhibition.

CASE II.

R.T., 58, tailor, admitted 19th August, 1901, complaining of cough and wasting away. He had been troubled with cough for two years previous to admission. Occasionally he had slight haemoptysis. He was a poorly developed man, very emaciated and anaemic. His complexion was muddy. Finger ends were clubbed. On admission, he weighed 8 st. 5 lbs. His temperature was subnormal. His previous health had been good. There was no insanity in the family. His mother died of "consumption" and his father and three sisters were alive and healthy. The whole of the right lung and upper lobe of left were infiltrated. During his stay in hospital, the lung condition quietened down. The temperature, excepting for an occasional rise of a few degrees for a day or two, was subnormal. The pulse varied between 70 and 90 per minute and kept a fair tension. He/

He lost 5 lbs. in weight whilst in hospital. The urine contained no albumen.

He was an extremely quiet man and at no time until one year after admission, did he show any mental change. Then one day he suddenly got out of bed and wandered aimlessly about the ward. He was very restless and continually attempted to get out of bed. He was noisy and talked incessantly. He became very abusive and showed a great dislike to one or two of the patients. He was obscene and used threatening language. There was complete loss of memory for recent events. He did not know where he was and did not recognise the nurses. His memory for more remote events was good, for he knew the sister of the wards who came from an hospital where the patient had been treated three years previously. He steadily got worse. He talked incoherently and became suspicious and more and more restless. He became so noisy and threatening, that it was deemed advisable to send him to the Asylum. He died in the Asylum six weeks later. Death occurred three years after the commencement of his illness. The lung lesion was of a very chronic type.

CASE III./

CASE III.

T.M., 37, carter, admitted 15th September, 1902. He complained of cough and shortness of breath. His illness definitely dated back six weeks before admission. His sputum had occasionally been blood streaked. He had got very weak and lost weight rapidly. He was a tall, well developed man, fairly well nourished - skin was very dry.

His family history was satisfactory. He had had no important illness, previous to his present ailment. He had been a heavy drinker. His right apex was consolidated and the lesion was fairly active. There was no albumen in the urine. His bowels were very constipated throughout the whole of his illness. Fourteen days after admission, he had slight haemoptysis repeated at intervals.

At this time, he became hypochondriacal. His whole mind was centred on himself. Every day he complained of pains and tenderness all over, but especially in his limbs and chest. Later on, he became very restless, getting out of bed and wandering about. He talked incoherently and had hallucinations of vision. His general condition got worse and he lost flesh rapidly.

His mental condition improved somewhat, but he gradually/

gradually got weaker and weaker and died nine weeks after admission.

This case ran an extremely rapid course, death occurring four months from the beginning of his illness. The temperature remained subnormal throughout.

The pulse rate was persistently increased, varying from 90 to 120 per minute. It was regular, but of low tension. The early termination after the onset of mental symptoms was more than one could have anticipated.

REMARKS:

The most striking feature in these cases was the rapidity towards a fatal termination, after the onset of the mental symptoms. The chief symptoms in the cases were, suspicion, irritability and restlessness. At times, they occasionally had lucid intervals.

Cases 1 and 2 were characterised by hilarity and buoyancy, whilst case 3 was depressed and hypochondriacal. The lesion in any of the cases was not of an acute character, and it was more or less quiescent in all. The temperature did not rise, nor the pulse rate increase with the onset of mental symptoms, but emaciation became more pronounced.

Anaemia/

Anaemia was a striking feature in all three.

In the first case there was a history of mental disturbance in the family; in the others, there was no evidence of this.

There was no discoverable exciting cause in any of them, although in Case III. it was significant that the mental change followed immediately after the onset of haemoptysis. No doubt, grave visceral disease so lowers the stability of the mind that mental aberration arises. In this case, pain and superficial tenderness were complained of. These perhaps were reflected from the affected viscera and may have been the direct causes of the mental aberrations.

The prognosis would appear to be unfavourable, but according to Dr Clouston, 30% of the cases recover.

#### TREATMENT:

The treatment adopted is based on the open air system as practised in sanatoria. As is well known, the principles upon which this system rests are:-

1. The maximum of fresh air and sunlight.
2. Liberal feeding or stuffing.
3. Regulated exercises.
4. Close medical supervision.

It may be said at once that our treatment falls short of these; nevertheless, I think the results are better than those obtained in hospital or general practice by treating such cases on the old protective system.

As far as possible, the patients are up and out of doors. Only cases with a temperature above 100° F. are kept in bed. The greater part of the day is spent in the open air, but unfortunately, the space at the patients' disposal is limited and surrounded by buildings, so that they are but little exposed to sunlight. Revolving shelters are provided as a means of protection against wind and rain.

Fortunately, wards are set apart for the phthisical patients, consequently there is no risk of infecting other patients.

The wards are old, badly constructed and so situated that very little sunlight enters them. The windows of the wards are kept widely open night and day, care being taken to avoid draughts. Heating is by means of fires, so that it is impossible to keep the wards at an equable temperature.

It is a point of some interest to note how little there is to be feared from cold or chills. New patients get no harm from at once adopting an open/

open air regime, and they very soon get accustomed to it.

#### FEEDING:

Three meals are given daily, with long intervals between them, during which no food is allowed.

The meals are plain and consist of the following:-

Breakfast:- Porridge, 3 oz. bacon, 1 egg, 7 oz. bread and 1 oz. butter, 1 pint of tea.

Dinner:- Boiled or roast meat, boiled or roast mutton, chicken or rabbit, six times weekly; 4 to 6 oz. daily; fish once a week. Potatoes, 8 oz., fresh vegetables daily; pudding, rice or sago, 6 oz.

Tea: Bread, 7 oz., butter 1 oz.; milk, 1-2 pints daily. Malt and Cod Liver Oil, 2 teaspoonfuls thrice daily.

No attempt is made at stuffing, but patients may have more than the above if they have the desire. On the above diet, some of the patients gain from 1 to 2 lbs. weekly; so that in two months, there may be a gain of a stone or more.

Regulated exercise and close medical supervision could not be carried out.

Systematic treatment with drugs, with the exception of urea (see urinary system) was not carried out. Of course, symptoms were treated when they arose.

How much does one accomplish in their treatment? Bearing in mind the very advanced cases presenting themselves at a workhouse hospital, too much need not be hoped for in treating such cases. Many of the cases are in a stage far beyond that in which treatment is of much service. Such patients are made more comfortable, many of their distressing symptoms are relieved and the duration of life prolonged. Less advanced cases do benefit and some of them are fit to return to work. By coming under the influence of a better diet, better hygienic conditions and a regulated life, the lung mischief may be stayed. But in many, a change to their former life, with its bad influences, soon reduced them to their old state. A recrudescence generally occurs and usually runs a rapid course. One has seen the most marked changes in a patient returning to hospital after an absence of a few weeks. The disease would appear to run a more rapid course than before. It is remarkable how much improvement such patients undergo time after time on their return to hospital. Perhaps it is that their constitution gets accustomed to the debilitating influences to which it is subjected; but the change from hospital treatment to their former/

former life is so great that their resisting power is suddenly diminished. The chief object of treatment, therefore, must be to limit as much as possible those factors which reduce the normal resistance of the tissues.

The results, even in the worst cases, are such as seem to justify the method of treatment adopted. The disappearance of the most distressing symptoms and the return of comparative comfort are striking. I cannot do better than individualise some of the more evident results.

#### 1. ALIMENTARY DISTURBANCES:

One of the commonest complaints on admission is loss of appetite, but after a stay of a few weeks, this frequently disappears. This I look on as a good prognostic sign. Digestive discomfort and other alimentary disturbances, which form distressing features in most cases under the old system of treatment, are reduced to a minimum. Measures directed to the stomach are seldom necessary.

#### 2. RESPIRATORY SYSTEM:

Of special interest is the infrequency of haemoptysis. When haemorrhage did occur, it was usually scanty and not often repeated. In only one case of the series the haemorrhage was persistent and intractable/

intractable. Thirty-six of the patients gave a history of haemoptysis, yet only 7 of them showed it during their stay in hospital, which varied in the majority of cases from 6 months to 2 years.

Cough and expectoration lessen similarly, but of all the symptoms, cough showed the least improvement.

### 3. NIGHT SWEATS:

In no one symptom is the change so striking as in this. The majority of cases give a history of night sweats, yet after a short stay in hospital, this symptom is rarely complained of. It is true that in a number of the cases this symptom persists, but these are usually very advanced, and it may be their vitality is so much reduced that they do not react to the treatment. The use of special drugs for the sweating was rarely called for.

### 4. TEMPERATURE:

In many of the cases, a noteworthy improvement of the temperature took place. In the majority of cases in which there was an improvement in the general condition, a corresponding improvement in the daily variation of the body heat took place.

It is remarkable that such a change can be brought/

brought about apart from drug treatment. No attempt was made to reduce the temperature by means of drugs, but in several cases, tepid sponging was resorted to and with good results. The temperature did not rise after the sponging and the patients were infinitely more comfortable afterwards. In one case, improvement only set in after having recourse to sponging. After the sponging was discontinued, there was no rise of temperature.

#### 5. CIRCULATORY SYSTEM:

In like manner, an improvement of the pulse can be noted. A change from the soft wavering pulse of phthisis to one of better tension, is one of the most striking phenomenon in clinical medicine. Coincident with the improvement in the <sup>quality</sup>~~rate~~, a pulse of 100 to 120 per minute will sometimes in a few weeks fall to 70 or 90 per minute.

#### 6. GENERAL CONDITION OF THE PATIENT:

The pale, careworn and haggard look is often replaced by the appearance of comparative health. The patients are brighter and happier.

They may steadily gain weight. This increase is not merely confined to the adipose tissue, but there is a real increase of muscular tissue. The muscular/

muscular tone correspondingly improves and the feeble, stooped gait is replaced by the erect attitude and a firm solid step.

#### 7. PHYSICAL SIGNS:

These correspondingly improve.

#### EXAMPLE:

F.G., 29, tailor, admitted on 14th January, 1903, complaining of cough and wasting away. He had had a cough for past eight months, but had only felt in poor health for five weeks previous to admission. At that time, cough became very troublesome and he felt tired whilst at work. He expectorated a large amount of greenish purulent expectoration. He had never had haemoptysis. His appetite was very poor and he perspired profusely at night time. He worked until 14 days before admission, since when he had been confined to bed. He was a heavy drinker and lived in lodgings. His previous health was satisfactory.

#### Family Health:-

Mother died of "Consumption".

Father " of Pneumonia.

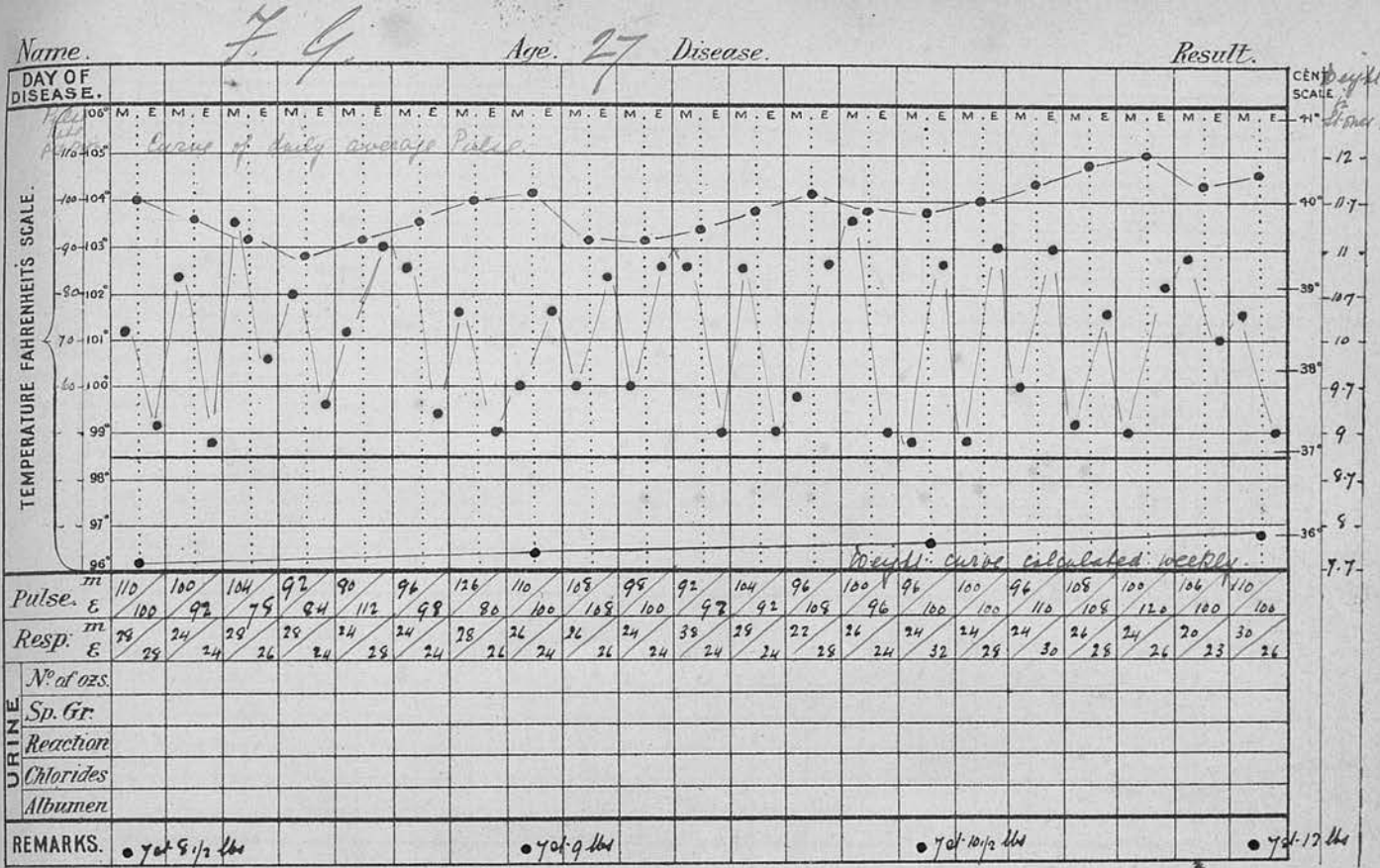
1 brother died of "Consumption".

Patient was very emaciated, but fairly well developed./

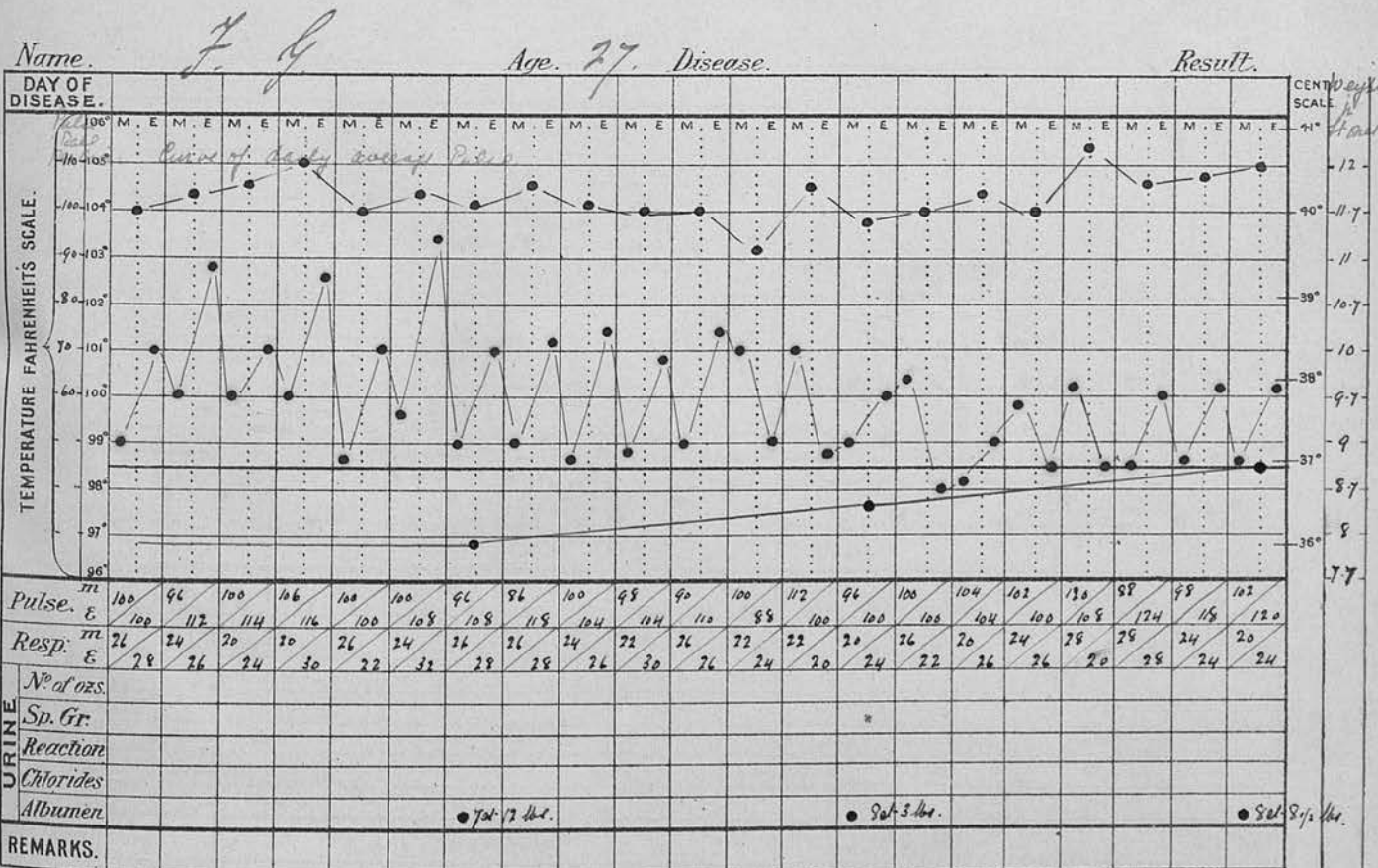
oped. Weight 7 st.  $8\frac{1}{2}$  lbs. Examination of chest revealed consolidation with cavity formation in the left upper lobe and infiltration of lower lobe. There were numerous sharp and bubbling accompaniments. The pulse was 104 per minute, regular, but soft and wavering. His temperature was  $100.4^{\circ}$  F.. At time of admission no very favourable prognosis was formed. For some time after admission, the temperature was of the reverse type, reaching to  $103^{\circ}$  F. in the mornings. The pulse had increased in frequency and night sweats became very troublesome. His appetite was fairly good, but he lost  $\frac{1}{2}$  lb. in weight. On the whole, the patient seemed worse. Three weeks after admission, he was tepid sponged daily. He immediately began to improve. The daily oscillations of temperature were less marked, night sweats disappeared and the patient's general condition seemed better. The temperature continued to fall, and its reverse character disappeared; by the 27th February, six weeks after admission, it did not go beyond  $99^{\circ}$  F. The pulse steadied down somewhat, but the improvement of the pulse was not so marked as the improvement of the temperature.

The appetite improved and weight was gained at the/

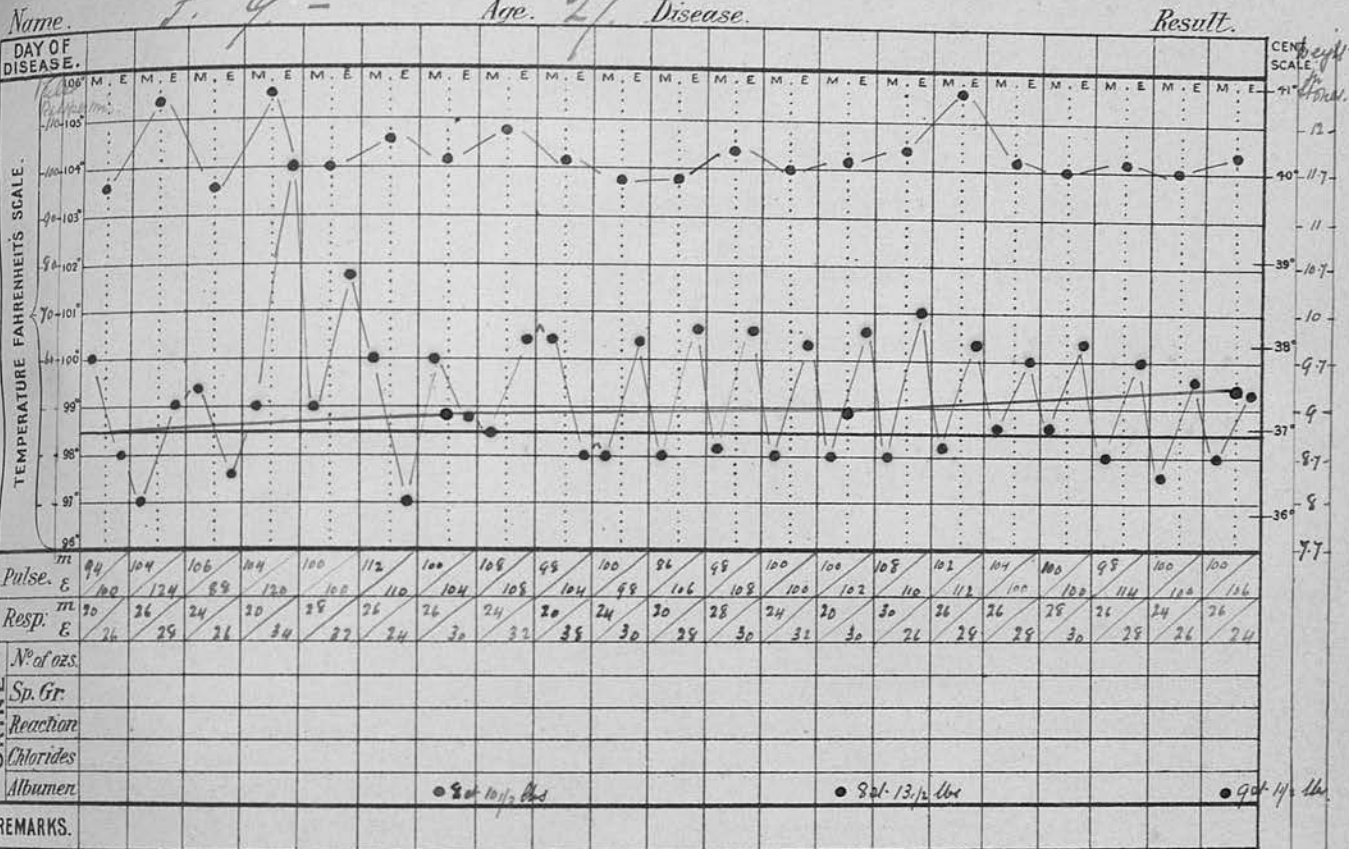
CLINICAL CHART OF TEMPERATURE & C.



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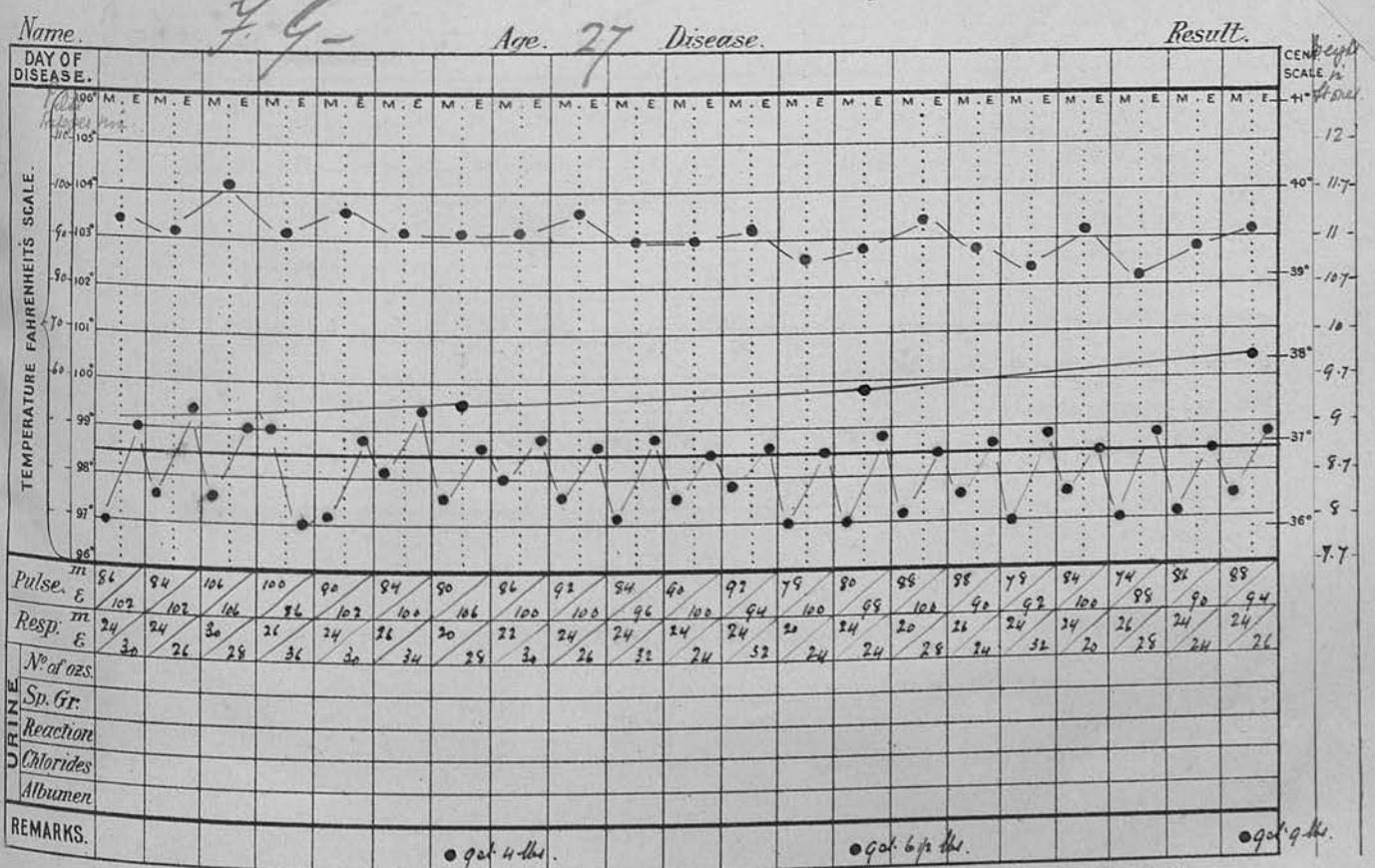
CLINICAL CHART OF TEMPERATURE & C.



(Observations taken at \_\_\_\_\_ A.M. and \_\_\_\_\_ P.M.)  
 For Memoranda of Treatment, see back of Chart.)

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the rate of 2 lbs. weekly. His weight was then 7 st. 12 lbs. Four days later, he had a little pleurisy, which caused the temperature to rise, but in 18 days, it was down to 99° F.

On the 4th of May, the following note was made. Patient has greatly improved. In 8 weeks has gained 16 lbs. Cough practically gone, expectoration scanty. No night sweats. The temperature is about normal. Pulse has improved, the tension is better and rate rather less. There are a few crepitations over left lung. There has been no extension since 1st April.

10th May:

Patient is still improving. Well nourished. Still gaining weight. Now weighs 8 st. 13½ lbs. Pulse less frequent. Appetite good. Patient says he never felt better. He has a ruddy complexion and looks the picture of health.

He is still under observation. The accompanying charts show the improvement in the temperature, pulse and weight.

Such a case shows that real benefit follows the mode of treatment adopted, yet it falls very far short of that carried out in well known sanatoria./

oria. Unfortunately, the majority of the cases in workhouse hospitals do not react so well to the treatment, but this is because of the very advanced class of cases met with.

Can the same treatment be carried out at the home of a patient? It can, and with as good results, possibly better because the cases can be got at an early state of the disease. Provided there is a sufficient appreciation of what is necessary and a sufficient determination to carry this out at all cost, good will result from this home treatment. The difficulties in carrying out such treatment are:-

1. Other members of the family have to be considered, especially in winter.
2. Insufficient accommodation.
3. Want of some one at home with the authority sufficient to insist upon rules laid down by the doctor to be observed.
4. The social position of the patient may be such that the hygienic and dietetic measures so essential may not be at his or her disposal.

PHTHISICAL PATIENTS UNDER THE POOR LAW.

The total number of medical cases admitted to this Hospital for the year ending June 30th, 1902 was 1132., Of these, 154 were cases of Pulmonary Tuberculosis - 120 males and 34 females; 56 of them died, giving a percentage of 36. This high death rate is due to the large number of advanced cases, and any appreciable diminution in the number of fatal cases cannot be hoped for until there is inaugurated a system of voluntary notification of the disease and a systematic dealing with the infected areas of the city authorities.

The only course open to Guardians, until recently, was to send their phthisical patients to their infirmaries. ; Where the amount of air space allotted to them, though, perhaps ample for ordinary patients, is entirely insufficient for persons suffering from Tuberculosis of the Lung and only in a very few of them is precaution taken to separate Tuberculosis from non-Tuberculous patients. By such a course of treatment not only is the recovery retarded, but actually prevented, whilst his presence in the wards inhabited by non-tuberculous patients/

ents is also a positive danger to the latter. I admit that, supposing we could train phthisical patients to exercise every possible care with regard to expectoration, the danger to other patients would be more theoretical than real. But we have to remember with regret that to a large extent, we are dealing with people who have not been in the habit of regulating their lives, have not taken the greatest possible care of themselves and are not likely to have too lively a sense of their duty towards their neighbour.

Should such patients expectorate about wards or grounds where non-tuberculous cases congregate, the latter run a very great risk of contracting a disease from which up to that time they had been free.

Hence it is obligatory upon those having the control of public institutions into which tuberculous patients are received to arrange for the complete separation of them from other inmates. This, fortunately, is now being done in many of the workhouse hospitals by having special wards for tuberculous patients. Dr F. R. Walters in his work "Sanatoria for Consumptives" says: "Every victim of unarrested phthisis is a possible focus for the dissemination/

dissemination of the disease. He is also a burden to his family, or to the state, or both, and if the bread winner, plunges those dependent upon him into serious pecuniary and social difficulties."

Remembering these facts and remembering also that in hospital, the chances of recovery are not very good, it is urgently desirable that other means for the recovery of the patients' health should be sought. Surely it would pay Poor Law Guardians to take steps to preserve the lives of such cases, who are in many cases the bread winners of families, and thus prevent those dependent upon them from permanently becoming a burden upon the rates.

For this object, the erection of Sanatoria are pre-eminently desirable. Fortunately, a considerable number of Boards of Guardians have resolved to erect Sanatoria, and I am glad to say the Bradford Board of Guardians have in course of erection, a Sanatorium suited for 40 patients.

The chief obstacle to the successful treatment of pauper patients will be owing to the difficulty of getting the cases in as sufficiently early stage to yield to treatment. In a large proportion of instances, by the time the people sought the shelter of the Poor Law, their condition will be so hopeless that to send them to Sanatoria would be fruitless/

less expense. The only course with such cases is to let them linger out their lives in the most comfortable method possible, at the same time taking the most elaborate precautions to prevent them becoming centres of contagion amongst non-tuberculous patients. What is necessary is a careful selection of suitable sanatoria cases. This could be done by the out-door medical officers.

What advantages are likely to follow on such treatment:-

1. It will have a tendency to lessen pauperism. At present, patients suffering from Pulmonary Tuberculosis go into workhouse hospitals, where they are located amidst unsuitable surroundings and are not subjected to a course of treatment which might be beneficial in their cases. The result is that they are not prepared for a return to ordinary life and active work; and if they do not die speedily in the Infirmary, they linger without a hope of recovery and on their decease their wives and children are left to the care of the State.

By adopting modern methods of treatment, we not only save lives, but preserve to the community and to their own immediate families, persons capable of doing useful and remunerative work.

2. The educational advantages are also important. Rigid rules are laid down for the conduct of the lives of the patients, and they are taught to regulate their lives in the best possible way. The result would be that when they left the establishment, they would spread amongst the community enlightened views as to the manner in which phthisical patients or patients with a tendency to tuberculous disease should conduct themselves in their own interests and in the interests of the community. How far these advantages will be realised remains to be seen.

The sole aim of the treatment advocated is to ensure the return of the patient to the normal standard of health and thereby render him capable of doing work. To achieve this, it will be necessary for him to live a physiological life. Any departure from this will result in the patient going back to the old state or further. The risk of such a condition of affairs will be very great amongst paupers, because by force of necessity, many of them are compelled to go back to their former life. The sanatorium treatment must be carried out as far as possible by these patients after leaving the institution, hence the desirability of some system of homes or colonies/

colonies for these cases. Dr R. W. Phillip urges the adoption of colonies which, he says, could be made the training school for a variety of out-door occupations, which, after a due probationary period, would come to be the means of a livelihood to the patient.